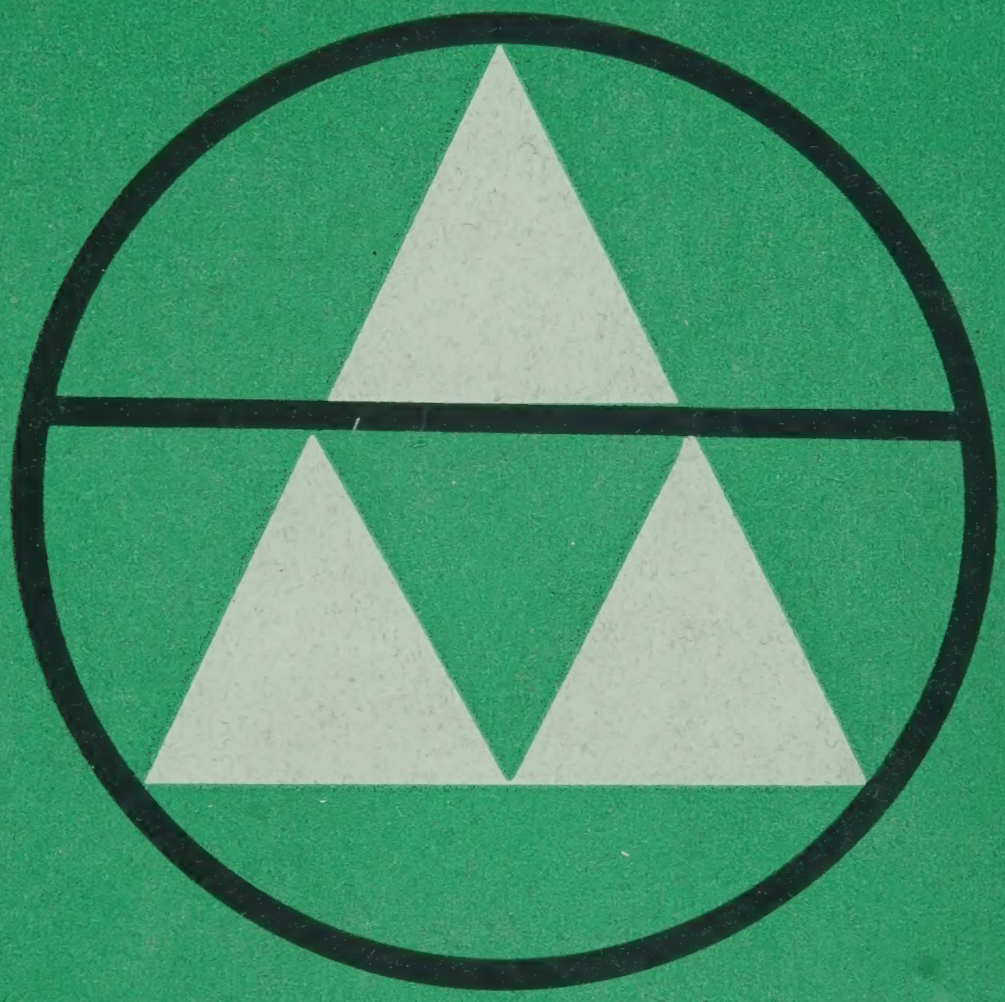
DISEASES OF FREE-LIVING WILD ANIMALS



FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

DISEASES OF FREE-LIVING WILD ANIMALS

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by

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FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

Rome 1962

Printed in Italy

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INTRODUCTION

Until about thirty years ago, little attention was paid to diseases of free-living wild animals, apart from a few of obvious importance to domestic animals or man. It was generally assumed that wild animals were healthier than their domestic counterparts and that little or no epidemic disease existed in such populations.

Mainly as a result of certain small wild mammals being adopted for use as experimental animals in research laboratories, information began to accumulate concerning mortality in those populations, but it was not until 1931 that Elton, in his classic monograph, described the existence of a large unexplored field in the study of wildlife diseases, and urged for further investigations to be made.

The link between the occurrence of disease and the marked periodic fluctuations in some wild populations is now well recognized, although many other factors, such as climate, availability of food and stress are also sometimes involved. Increased attention is now being paid to this subject throughout the world, and the creation of the Wildlife Disease Association of the United States is an excellent example of this growing interest. There is undoubtedly a belief among research workers in the veterinary and public health fields, quite apart from those interested in ecology, that this will be of considerable interest for further study. In the present publication, no attempt has been made to deal with the lower forms of animal life or with helminthiasis and the numerous ectoparasites recorded from wildlife, as it was felt that all these fields were worthy of separate consideration in due course. Accordingly, this survey has been confined to a consideration of disease in free-living mammals and birds brought about by bacteria, viruses, fungi, protozoa and rickettsiae, paying particular attention to those conditions communicable to man and/or domestic animals. A small section on tumors is also included, although authentic information on this subject was difficult to obtain.

Various groups of individuals are affected by this problem of disease in wildlife. Naturally, it has always been a matter of concern to those interested in game preservation, and now that game is frequently considered in the nature of a farm crop, the matter seems to be of increasing importance. It is perhaps worth recalling that many years ago "grouse disease" in Scotland was considered to be of sufficient importance to warrant the setting up of a special committee to inquire into this problem. Another enterprise, namely fur trading, has always been much concerned in the fluctuations of animal populations. The snowshoe rabbit inquiry conducted by Oxford University over a period of many years has shown the importance of enzootic disease as a limiting factor in the number of this species and consequently a reflected variation in the number of fur-bearing predators from season to season. The returns of the Hudson Bay Company over a prolonged period do in fact show some very interesting trends in these populations, and it is possible to predict good and bad years by a careful study of these statistics. Naturally, the student of ecology is interested in these phenomena but many of the fluctuations, especially in the smaller mammals, still remain to be solved.

The establishment of nature reserves and national parks in many countries emphasizes still further the need for knowledge of disease in free-living species and its effect not only on the animals themselves in semiartificial conditions but also on incontact domestic animals and man.

When diseases of wildlife are considered from the public health angle, numerous important associations are found — these cannot be ignored, especially with the ever-increasing ease and rapidity of transport between countries. Moreover, the reclamation or development of large tracts of virtually uninhabited land in different parts of the world where wild fauna may readily act as reservoirs of infection, such as leishmaniasis, also constitutes an important problem.

Needless to say, one of the most important points to consider is the close connection between diseases in wildlife and domestic stock. For the effective control and eradication of disease, the veterinarian must know the possible reservoir hosts for the various infective agents. It would, for example, be exceedingly difficult to eradicate *Brucella abortus* infection from cattle if the disease were present in wild animals in the vicinity. This has already to some extent been proved in Yellowstone National Park in the United States, where repeated fresh outbreaks of disease in domestic cattle have been associated with infected free-living

elk in the vicinity. The close affinity which exists between disease in game and domestic stock is perhaps best demonstrated by the situation in Africa, where it undoubtedly constitutes a major problem in some territories. From the agricultural point of view, the introduction of myxomatosis has played a vital part in the control of rabbits in Australia, France, the United Kingdom and elsewhere; undoubtedly, this one disease of wildlife has already changed the whole economy of farming in the countries concerned and, for that matter, other industries as well.

From the above, it will be realized that this subject, in one way or another, affects many sections of the community — the farmer and his veterinary surgeon, the patient and his doctor, not to mention the student of ecology and the epidemiologist.

In this monograph, no claim is made to having surveyed the entire literature in every language; there is a good deal of duplication in the different journals; much of the information is not complete and consequently the facts selected were those considered to be of most value to the general reader and to serve perhaps as a guide into the literature for those wishing to specialize in a particular subject. Consequently, the majority of references are concerned with recent work. Other references can readily be obtained by perusal of those cited. Finally, the mammals and birds are referred to by their "popular" names. This has been done because the writer's experience has been that apart from a limited few, trained in precise classification, the majority of medical or veterinary workers are unacquainted with the finer details of zoological nomenclature. In most cases, those who wish can obtain the precise scientific names from the original publications.

Much of the subject matter in this study has already appeared in the form of an FAO working document (*Animal Health Branch Monograph* No. 1, 1960) in one language, English, and without illustrations; this was primarily for circulation to veterinarians and others interested in the subject within the Organization.

It would be impossible to list all those who have helped the author by supplying material, photographs, advice and encouragement, but the following institutes and individuals are worthy of special mention: Imperial Chemical Industries Game Research Station at Fordingbridge; Wellcome Laboratories of Tropical Medicine, London; Ministry of Agriculture, Fisheries and Food, Central Veterinary Laboratory, Weybridge; Galli-Valerio Institute, Lausanne; State Serum Laboratory, Copenhagen; Research Institute of the Finnish Game Foundation, Helsinki; Pasteur

Institute, Paris; the State Veterinary Bacteriological Laboratory, Luxembourg; the University of British Columbia; and the Fish and Wildlife Service of the United States; the Hon. Miriam Rothschild, Professor R. A. Willis, Professor M. Christiansen and Professor I. McT. Cowan; Dr. A. Q. Wells, Dr. R. E. Rewell, Dr. J. R. Wadsworth, Dr. C. M. Herman, Dr. M. K. Lund, Dr. H. Jacotot, Dr. J. C. Broom, Dr. C. A. Hoare, Dr. G. Bouvier, Dr. C. Gottal, Mr. M. Helminen, Mr. P. K. C. Austwick, Mr. A. D. Middleton and Dr. P. Clapham.

The editors of the following scientific journals kindly allowed the reproduction of certain photographs: *Journal of Hygiene*, *Journal of Comparative Pathology*, and *Journal of Animal Ecology*.

In addition, the author would like to express his sincere thanks to Dr. W. S. Gordon, Director of the Agricultural Research Council's Field Station, Compton, who afforded every facility for this work, and to Miss F. B. Sutherland and Mrs. M. Walters, who gave valuable assistance in the compilation of data.

1. DISEASES DUE TO BACTERIA

Staphylococcal infection

Isolated instances of staphylococcal infection have been described in several countries. In Italy, a pyemia in wild hares caused by staphylococci was recognized in 1932 by Spinelli and Penso; a further report from the same country by Corsico and Poggi (1953) drew attention to a form of septicemia in the same species in the Lombardy district, the causal agent, according to the authors, being *Staphylococcus albus*. Bouvier, Burgisser and Schneider (1954) mentioned the occurrence of the infection in hares in Switzerland associated with a variety of lesions, from which *Staphylococcus aureus* could be isolated. They also pointed out that other workers in Switzerland had found *Staphylococcus albus* to be the causal agent in some cases.

In the United Kingdom, McDiarmid (1955) recorded the occurrence of an idiopathic staphylococcal pyemia in a wild brown hare, the infection being apparently unrelated to any external mechanical injury. The hare was found alive on the Berkshire Downs and the general impression was that it had been affected with some form of paralysis rather than toxemia. Numerous dead hares had been noticed in the area by different observers during that particular season (1950), but unfortunately none had been examined. The principal lesions involved the heart (Figure 1) and kidneys. There was no evidence of any lesions impinging on the central nervous system nor of external damage. The culture recovered from this particular hare was *Staphylococcus aureus*. It was hemolytic ($\alpha\beta$), coagulase positive, and eventually was shown to belong to the 42D group by bacteriophage typing.

The strain was pathogenic for white mice, intravenous inoculation producing a typical pyemia. Sections of the heart, stained by hematoxylin and eosin and by Gram's method, showed the formation of typical abscesses in which cocci were numerous. This particular type of staph-

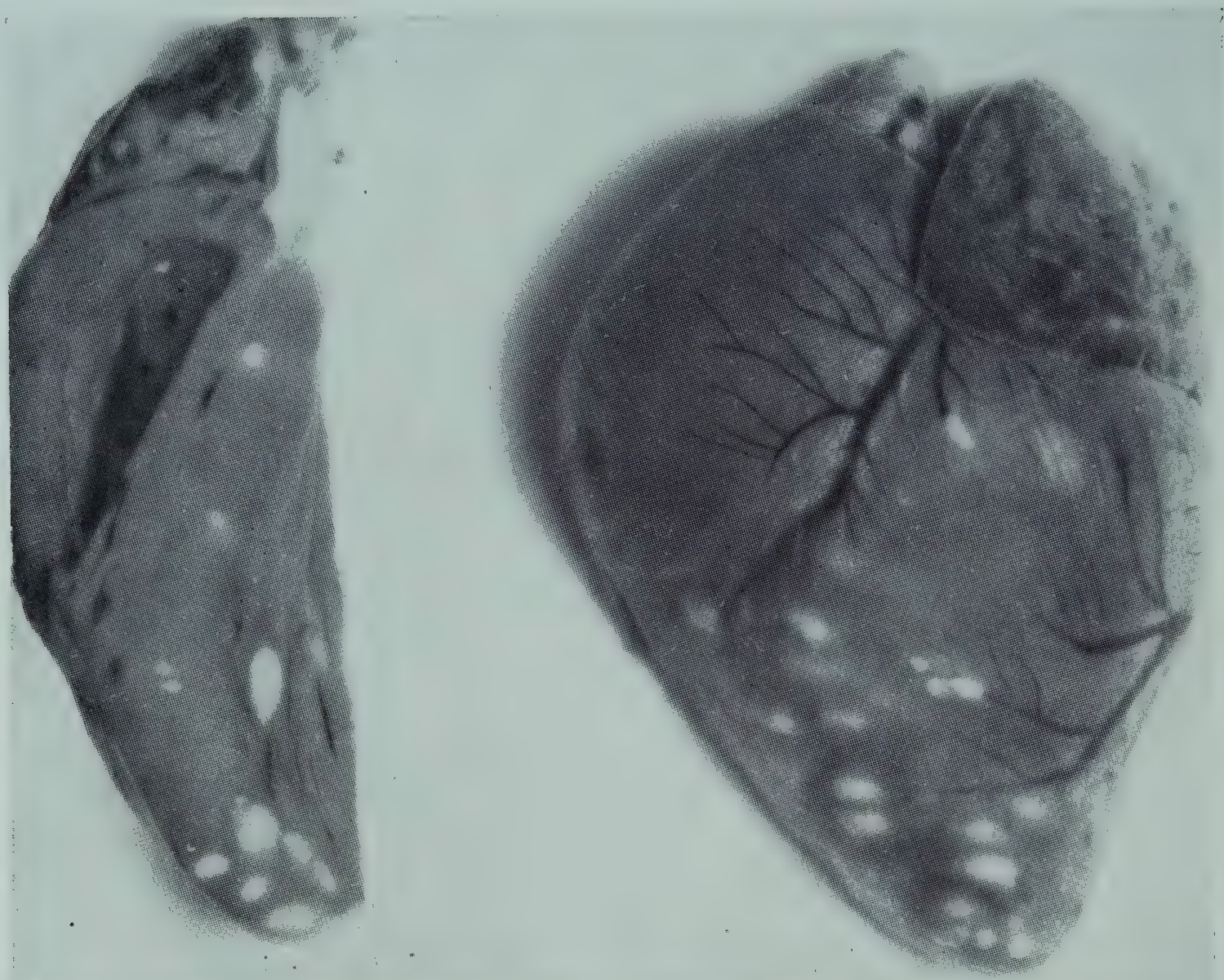


FIGURE 1. — Staphylococcal pyemia in a hare; macroscopic lesions in the heart. Courtesy *Journal of Comparative Pathology*

ylococcus has been shown to be associated with disease in man and domestic animals (Smith, 1948), and is probably one of the commonest strains of staphylococci associated with mastitis in the dairy cow. This hare was, therefore, harboring an organism of some importance to veterinary and public health. Mastitis has also been observed in a wild rabbit in the United Kingdom (McDiarmid, 1944). A typical chronic mastitis, demonstrated macroscopically and microscopically, was present and a culture from the affected tissue was typed as *Staphylococcus aureus* on the basis of biochemical, serological and hemolytic tests. It was a typical coagulase +, $\alpha\beta$ hemolytic strain, pathogenic for mice by intravenous or intraperitoneal inoculation. Unfortunately, it was not possible at that time to identify the strain accurately by the bacteriophage technique. Apart from the mastitis, the rabbit appeared to be quite healthy when shot. Jennings (1954) has described the finding of coagulase +

strains of staphylococci from a hawfinch and a green woodpecker, found dead in the United Kingdom.

The only lesions observed were a few petechiae in the heart and congestion of the viscera. In both birds isolates were made from the heart blood, liver and bone marrow.

Streptococcal infection

Very few instances of confirmed streptococcal infection have been recorded in free-living wild animals. In the United Kingdom, Jennings (1954) recovered a type of streptococcus with the characteristics of Lancefield's group C from the great tit and the starling. From British hares in Berkshire, McDiarmid (1946) recovered a group D streptococcus which could not be differentiated from cultures obtained from fatal cases of acute endocarditis in Hampshire Down sheep grazing in the same area. The hares, which had been found dead, showed lesions of peritonitis and pleurisy. In this instance, it appeared that this particular wild species could harbor an organism which, under the prevailing conditions, could be considered potentially pathogenic for sheep. Mantovani and Ceretto (1953) also described the occurrence of streptococcal infection in hares in the Piedmont region of Italy.

Air sac infection (*Pseudomonas aeruginosa*)

In recent years, the author has found an increasing number of birds of the Corvidae group in the United Kingdom, with extensive lesions in the air sacs and, in some instances, the lungs. This disease is usually encountered during the summer months, especially July and August. The birds are usually emaciated when found dead, and a post-mortem examination reveals a chronic inflammatory condition of the lining membrane in one or more of the air sacs. The abdominal sacs appear to be most commonly affected. One or both lungs may also be involved, the lesion consisting of necrotic tissue surrounded by a zone of inflammatory cells. A bacteriological study of the organisms associated with this condition shows that a Gram-negative organism, *Pseudomonas aeruginosa*, appears to be the principal agent involved.

In some instances, other organisms may also be present; this, however, is to be expected in a disease affecting the respiratory system. The

carrion crow, rook, jackdaw and starling have been found to be affected, and it is likely that this list will be considerably extended by further inquiry and investigation. It is perhaps worthy of note that domestic fowls have also been shown to be subject to this particular condition.

Tuberculosis

A large proportion of papers dealing with disease in wildlife is concerned with the occurrence of tuberculosis, and a considerable amount of evidence is now accumulating to show that this disease may be present in several species of birds and mammals in many countries and must always be considered in any scheme for the final eradication of tuberculosis from domestic stock. So far as the author is aware, there are very few clear-cut records of disease in wildlife resulting from infections with the human type of *Mycobacterium tuberculosis* and, consequently, the disease in free-living mammals appears to be caused chiefly by bovine strains of the organism with occasional cases of avian tuberculosis. Unfortunately, many records of tuberculosis are completely devoid of precise details concerning the typing of the organism, and consequently the reader is left in some doubt concerning the exact etiology of the condition. Feldman (1938), in his classic monograph on avian tuberculosis, makes a plea for further investigation and precise identification of the strains isolated from wildlife, and this cannot be too strongly supported. In the larger mammals, the deer family have probably been shown to be more frequently affected than any other species. This is, again, due principally to the interest taken in their well-being by sportsmen in various countries, especially in Germany, where frequently facilities are available for careful post-mortem and further examination of any suspicious tissues. Thus records from Germany include a description of acute miliary tuberculosis in a roe deer by Platen (1939) and further cases in the same species by Hillenbrand (1940) and Schmidt (1938). The disease is also said to occur in fallow deer, and Witte (1940) described the disease in red deer in the same country. It is interesting to note that Bouvier (1947) appears to have recovered the human type of mycobacterium from a roe deer in Switzerland. Tuberculosis occurs in elk in Scandinavia. Hermansson (1943) and Christiansen (1931) encountered the avian variety in deer in Denmark. Further cases have occurred in antelopes in Africa (Robinson, 1953) and bison in America

(Hadwen, 1942). Cowan (1951), in Canada, mentions the presence of tuberculosis in buffalo, moose and elk under semiwild conditions in Alberta, and in buffalo and barren ground caribou under completely wild conditions, but unfortunately no precise information is available on the types of organisms involved.

In Europe, among other species, the chamois has been found infected with the bovine type by Bouvier, Burgisser and Schweitzer (1951), and Heidkamp (1939) found the infection in wild pigs. A seal has been found infected by Grini (1944 a) in Scandinavia, and Plum (1942) isolated organisms of the avian type from wild rats. Tuberculosis in rats has also been reported by Hulphers and Henricson (1943) in Sweden, and Bosworth (1940) in England. The two strains isolated by Bosworth were classified as bovine, and it is interesting to note that no gross lesions were present in those rats. In Africa, de Kock (1938) encountered a case in a kudu in Albany Cape Province. Strains isolated from kudu have been bovine in type. A human strain of *Myco. tuberculosis* has been recovered recently from a wild vervet monkey in Kenya.

The author has received information which indicates that tuberculosis may be encountered from time to time in red deer in Scotland and the southwest of England, but this is based purely on macroscopic examination, and no bacteriological confirmation is at present available.

In birds, numerous cases have been confirmed in various countries and it is perhaps convenient to consider them in different groups according to the country involved. One might have expected that many of these cases would have been found in predatory birds, i. e., those species which prey on other birds and which would naturally select those which were least able to escape attack. However, this is not so, and although some cases have in fact occurred among hawks, the vast majority have been confined to other birds, especially the granivorous species.

In the United States, the disease has been identified in hawks by Steinhaus and Khols (1942), and Beaudette and Hudson (1929) refer to its occurrence in the cowbird. Harshfield *et al.* (1937) examined a considerable number of crows, and tuberculosis was confirmed in several specimens. In Canada, Mitchell and Duthie (1929, 1950) showed the common crow to be frequently infected. The interesting feature of these cases was the intermediate characters of the organisms which they recovered; they appeared to be aberrant avian strains when examined culturally and biologically. A further interesting case in a crow was recorded by Beaudette and Hudson (1936) concerning the co-existence of a nema-

tode parasite with tuberculosis. A similar case of co-existence with nematode infestation was noted in the pheasant by Beaudette (1942) in the United States. A single case of tuberculosis in a Canadian widgeon was reported by Cowan (1941).

In Germany, avian infection has been reported in a buzzard by Kleinschmidt and Westphal (1950).

In Australia, the disease has occurred in the wild brown hawk (Rac, 1951) and in the gray teal duck (Sinkovic, 1954).

Plum (1942) has encountered the disease in a variety of species in Denmark, viz., the scoter, rook, pheasant, golden plover, kestrel, pigeon, gulls (various species) and sparrow. He considered that there was considerable risk of transmission of infection to domestic stock. Christiansen, Ottosen and Plum (1946) also encountered an acid-fast infection in wood pigeons which was not, in their opinion, typical avian infection. Masses of acid-fast organisms were present in smears but no cultures could be obtained on the media normally used for the isolation of *Myco. tuberculosis avium*. Grini (1942), in Norway, also found infection in the blackcock, and later in the capercaillie (1944 b). In Sweden, the infection has been confirmed in magpies and crows by Hülpers and Lillengen (1945). Bygyaki (1955) has recorded the avian infection in wild duck in the Congo.

In the United Kingdom, tuberculosis in birds has been recorded intermittently over a period of many years. Hammond Smith (1908) identified the disease in a kestrel hawk, and an infected green plover was found in Scotland in 1909 by Shattock *et al.* A blackbird was shown to be affected by Hare (1932). Unfortunately, many of the early cases were not investigated bacteriologically and the precise nature of the infecting organism was not determined. Hignett and MacKenzie (1940) examined a collection of various species of wild birds. They showed that the incidence of acid-fast infection was quite high in starlings and also demonstrated the organisms by biological tests, using excreta collected from below the roosts of these birds. The actual incidence of the disease varied from 2.8 to 4.8 percent, and they pointed out that the alimentary tract was principally involved, the vicinity of the caeca being a favored site. Cases of avian tuberculosis in a herd of pigs in Berkshire have been shown by the author to be closely related to the presence at the feeding troughs of large numbers of starlings and rooks, some of which were shown to be carrying the avian type of the organism. Harrison (1948) reported tuberculosis in a sparrow hawk, but unfortunately the

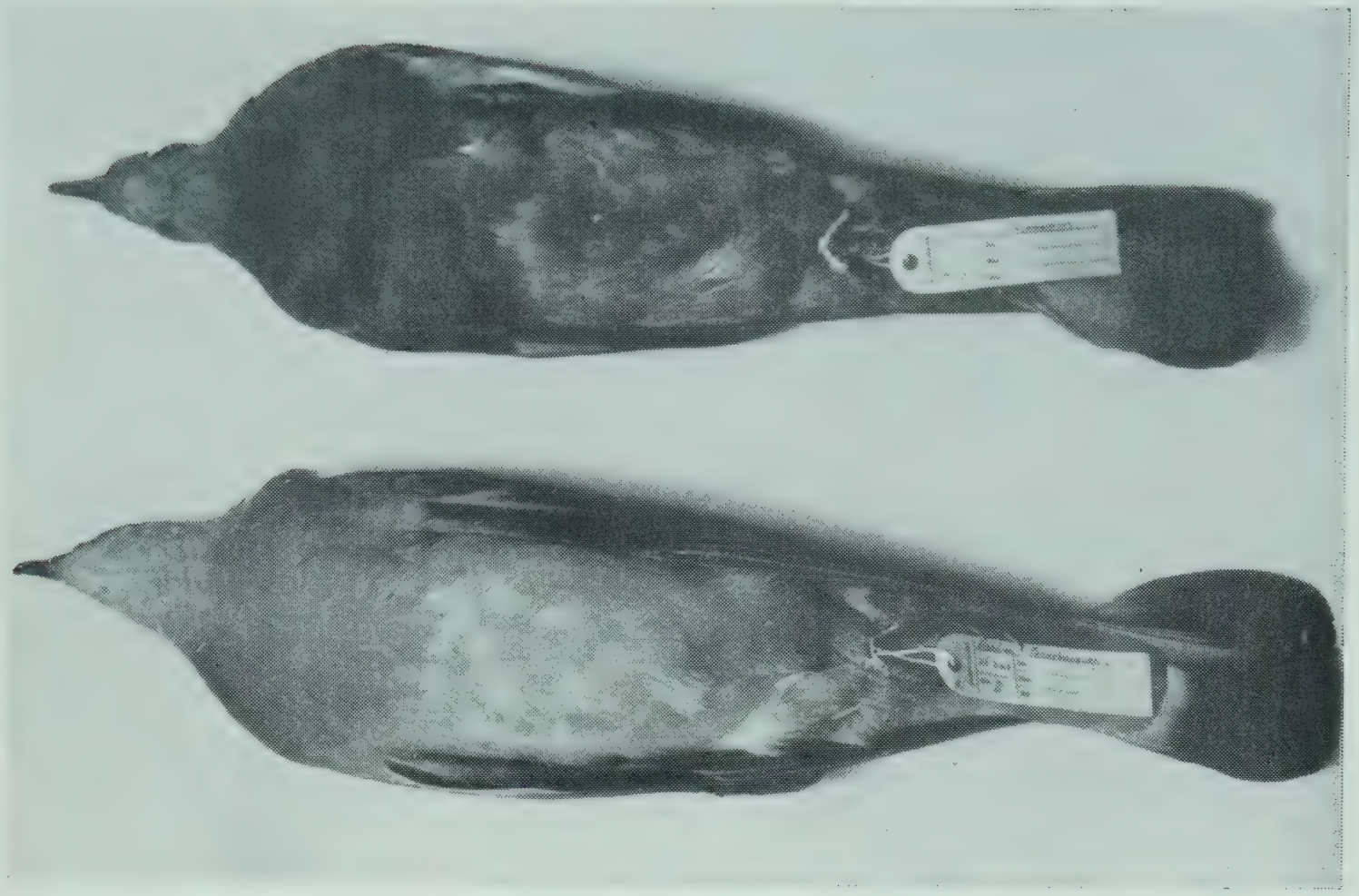


FIGURE 2. — Plumage change in a wood pigeon affected with tuberculosis. The affected bird is smaller and darker in color compared with the normal.

organism was not accurately typed, although the evidence strongly suggested it was avian. Other instances of the disease have been noted by Luke (1954) in blackheaded gulls, in the shag and in the widgeon duck (Harrison, 1955). Doyle (1943) confirmed avian tuberculosis in chaffinches and feral London homer pigeons. The author has encountered the disease in a variety of species, such as the pheasant, partridge, rook and starling, but the principal host for the mycobacterium in wildlife in the United Kingdom appears to be the wood pigeon (McDiarmid, 1948, 1954). In 1948, the cultural and biological properties of strains of *Myco. tuberculosis* isolated from wood pigeons were described in detail; a most interesting feature of the disease was the variation in color of affected pigeons (Figure 2). This was first noticed by the Hon. Miriam Rothschild and was considered to be similar to the pigmentation of the skin in Addison's disease in the human being. In wood pigeons affected with tuberculosis the adrenal glands are often involved, and it was thought that this might be responsible for the production of the bronze-green coloration of certain of the feathers, particularly the mantle. Frequently, the feathers have a matt finish instead of the normal bloom.

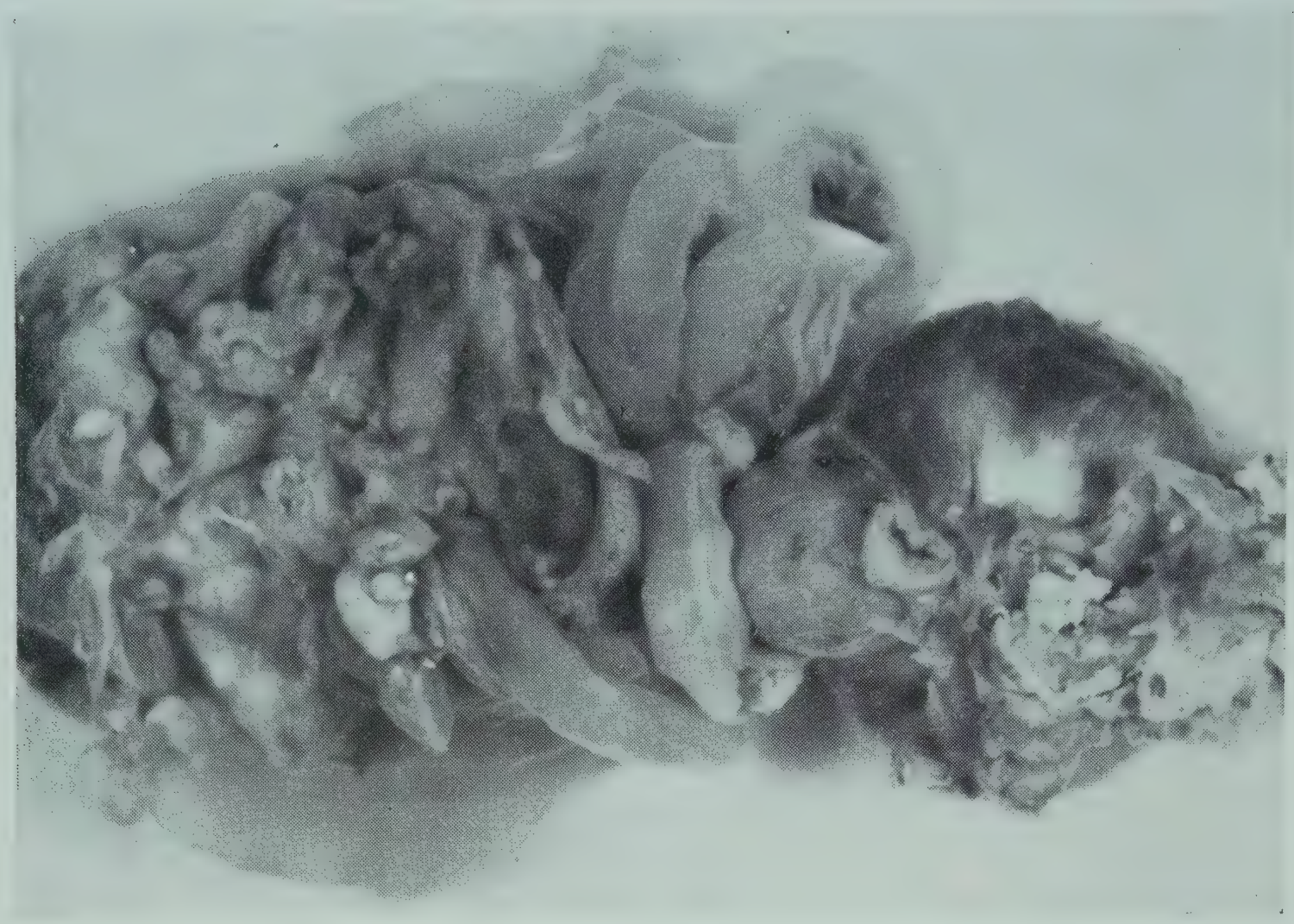


FIGURE 3. — Nodule formation in the abdominal viscera of a wood pigeon affected with tuberculosis.

However, this color change is not absolute, and perhaps only 50 percent of dark-colored, emaciated pigeons are, in fact, affected with avian tuberculosis. The incidence of the disease in wood pigeons varies from year to year (2 to 4 percent) and these infected birds must constitute a hazard to domestic poultry, particularly on free range. In areas where surface water is limited, the pigeons tend to congregate at the water troughs, and because of the extensive lesions in the intestine, they must readily contaminate the water and surroundings. There can be little doubt that many of the reactors to avian tuberculin found in young cattle after they are turned out on the pastures in early summer are caused by ingestion of this particular organism. This tends to make the interpretation of the tuberculin test difficult, especially when it is being utilized in an effort to diagnose Johne's disease.

When an affected pigeon is examined macroscopically, it is generally found that the disease has been of long standing (Figure 3), much proliferation of tissue has ensued, and wasting of the breast muscles is generally present. The general impression is that the host-parasite rela-

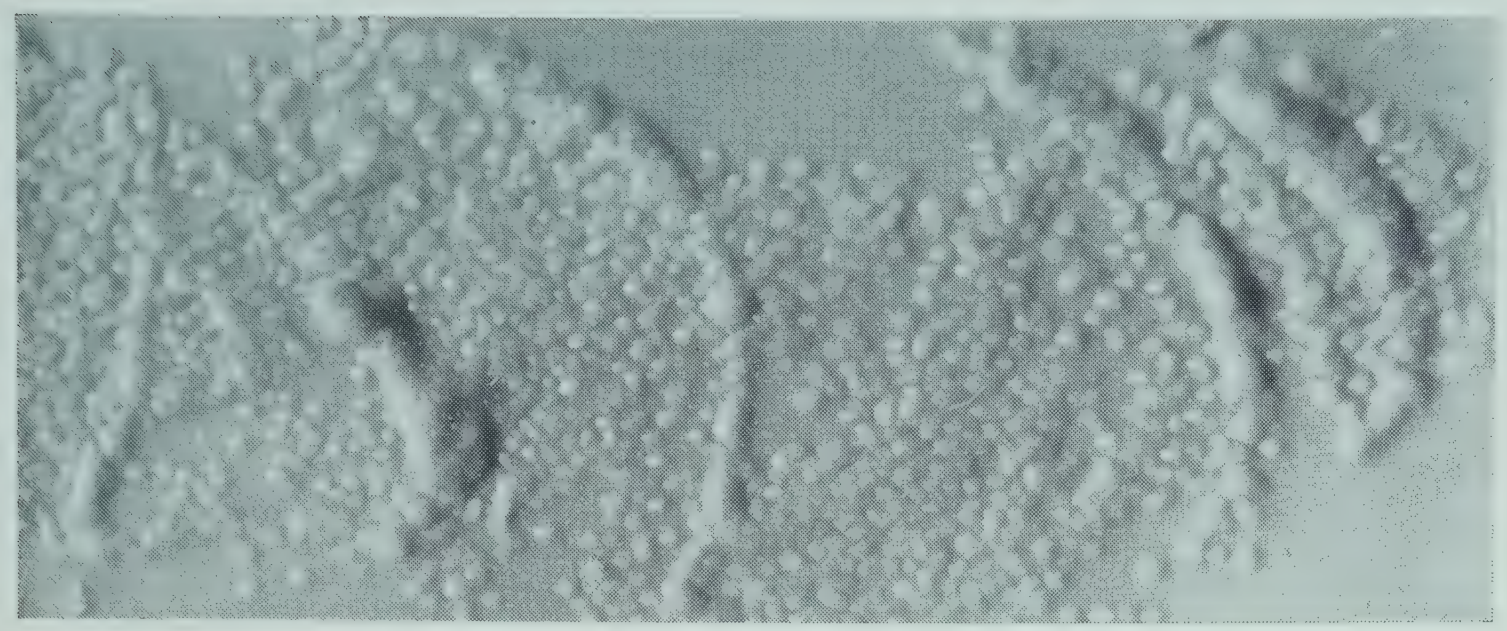


FIGURE 4. — Culture of a typical smooth strain of *Myco. tuberculosis avium.*

tionship is such that neither does the host get rid of the parasite nor does the parasite kill the host. This state of equilibrium is seen during the shooting of numbers of wood pigeons, because affected birds, apart from their darker plumage and small size, are indistinguishable from the normals in flight despite their long-standing disease.

Practically all the main tissues may be affected and smears from them show masses of acid-fast organisms. Cultures may or may not be obtained on media normally used for isolation of the avian type. If typical avian strains are present, growth on such media as glycerol-egg and plain egg is comparatively rapid and a smooth typical avian culture is obtained after about two or three weeks' incubation (Figure 4). However, from many pigeons no growth occurs on these media but, by using a medium for the cultivation of *Myco. paratuberculosis* (Johne's bacillus), such as Finlayson's or "lymph node" medium, Dr. A. Wilson Taylor succeeded in obtaining a slight growth in two or three months. Invariably, this growth is rough (Figure 5), and when the culture is inoculated into fowls or rabbits it fails to kill, although a sensitivity to tuberculin is produced. By passage, however, this culture will readily revert to a typical smooth avian type which will then behave in the normal manner. It is probable that this variant has arisen through a long sojourn in an unnatural host.

The epizootiology of tuberculosis in wood pigeons poses some interesting possibilities. The author's opinion is that the disease is enzootic in the pigeon population and that the infection is passed from pigeon to squab by the characteristic feeding habits of pigeons. Most of the

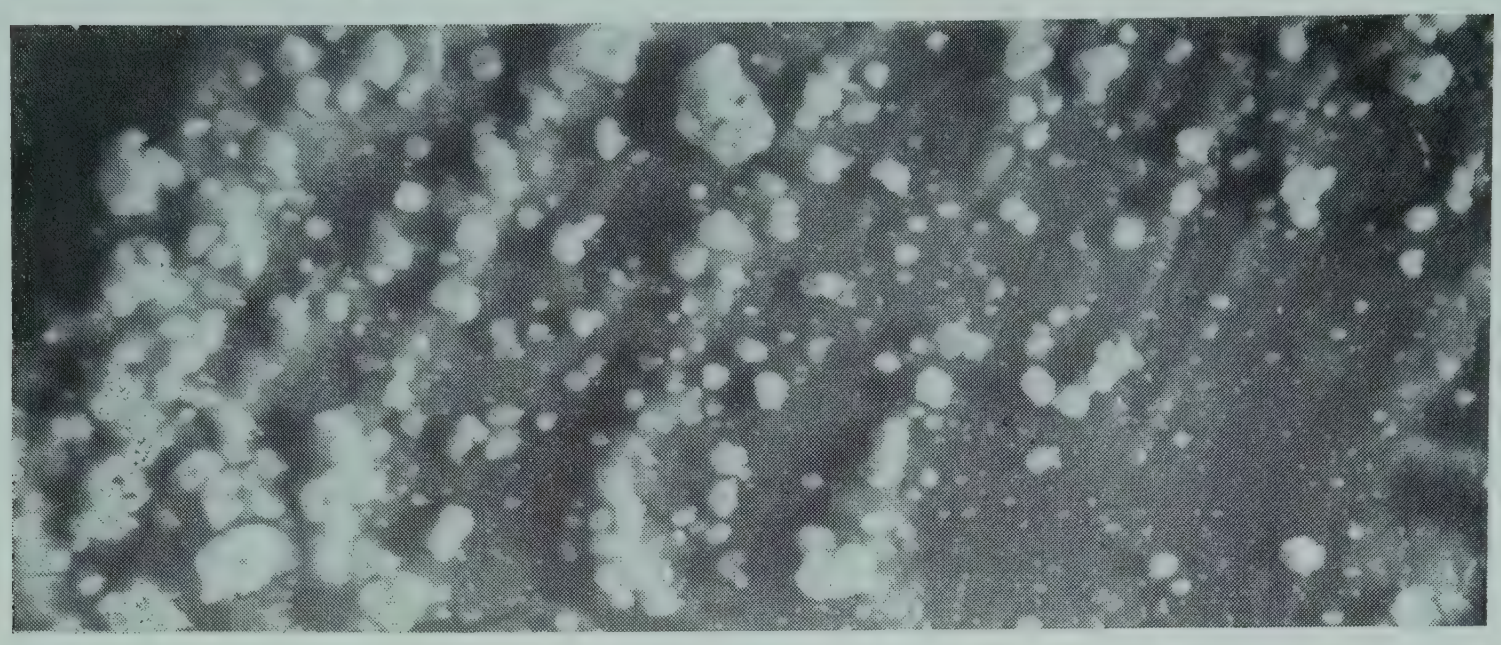


FIGURE 5. — Culture of an atypical "rough" strain of *Myco. tuberculosis avium* from a wood pigeon.

affected birds show involvement of the alimentary tract and the young squab has every opportunity of acquiring an early infection, either by direct ingestion from its mother's pigeon milk or from the highly contaminated nest. The effect of the disease on the pigeon population as a whole is probably not great, although it must be remembered that the majority of birds which eventually die from this disease are never found. In the author's experience of over 50 cases, only one bird has been found dead; all the others have been shot or caught alive. The majority of these birds have yielded the slow-growing rough type of organism which may well be identical to the so-called nongrowing strain previously encountered in Denmark (Christiansen, Ottosen and Plum, 1946). Many of the birds examined in the United Kingdom by the author during the winter months may, in fact, have been Scandinavian birds on migration.

Much further work is required on the problem of tuberculosis in wildlife, in view of the present attempts to eradicate the disease from farm animals in many countries. It would be futile to eradicate tuberculosis from cattle and then find breakdowns due to the presence of previously undetected foci of infection in neighboring wild species.

Tuberculosis in field voles

An unusual form of tuberculosis was first demonstrated in field voles by Wells in 1937, and since that date several reports on this condition have appeared in the literature (Wells, 1945, 1953; Wells and Wylie,

1954; Brooke, 1941). Bank voles, wood mice and shrews are also naturally affected.

A wild vole, caught in Scotland, was transferred as an experimental animal to a laboratory, and soon after admission was found to have a severe acid-fast infection. Subsequently, it was shown that this disease was common throughout the United Kingdom, sometimes assuming epizootic proportions — up to 65 percent of the field voles being diseased in certain instances. The violent fluctuation in the population of field voles every three or four years could justifiably be attributed in no small measure to this infective agent. Culturally, the organism behaved differently from the human, bovine and avian types of the mycobacterium, but serologically, it did not vary from the human and bovine types; a tuberculin prepared from the vole bacillus was qualitatively in no way different from these other two varieties. There was complete justification, therefore, for classifying this disease as a new type of tuberculosis, *Myco. tuberculosis* var. *muris*.

The organism was of low pathogenicity, very large doses being required to produce a progressive disease in the ordinary laboratory animals. Voles and certain allied species are, however, very susceptible. In the early studies of this new bacillus, it was shown that guinea pigs inoculated with this strain were subsequently resistant to infection with mammalian bacilli (Wells and Brooke, 1940; Griffith and Dalling, 1940), although complete protection was not established. However, some promising results were obtained in cattle immunized with the vole bacillus (Griffith and Dalling, 1940; Young and Patterson, 1949) and it has been used for the immunization of man. A similar, if not identical, organism has recently been isolated from the lungs of the cape hyrax, known locally as the "dassie" (Wagner, Buchanan, Bokkenheusen and Leviseur, 1958).

Johne's disease

Although cases of Johne's disease are known to occur in park deer, there is no precise scientific evidence that free-living deer are affected. However, the author has been informed by gamekeepers and foresters that a condition with macroscopically the same pathological pattern has been observed in fallow deer in certain forests in Wales and the south of England. No opportunity has occurred for bacteriological examinations to be made.

Rat leprosy

Rat leprosy is very similar to human leprosy and the infection can probably be transmitted to man (Marchoux, 1922). The first account of *Myco. leprae murium* was noted in 1903 by Stefansky at Odessa. About 5 percent of the rats he examined were affected. The disease is confined chiefly to brown and black rats. Reports indicate that it occurs in many parts of the world, e. g., in France (Marchoux and Sorel, 1912), Guinea (Leger, 1919), the United States (McCoy, 1913; Wherry, 1908), and in Japan (Ota and Asami, 1932). The incidence in these various places varied but was generally less than 1 percent.

Two principal forms of the disease are described, glandular and musculo-cutaneous, but there is no marked division between the two types. In the glandular form, groups of subcutaneous glands are enlarged, whitish and hard, but without nodules or necrotic areas. Microscopically, macrophages are numerous and packed with acid-fast organisms. Giant cells may be present. In the musculo-cutaneous type, emaciation and alopecia, especially on the head, are common findings and ulcers may be present. Frequently, the disease occurs in a latent form without clinical identification. Little is known of the precise way in which infection is contracted; it is probable that it occurs either by the nasal route or by superficial skin injury.

Melioidosis

Primarily, melioidosis is a disease of free-living rodents, but man can also be infected. In some respects the disease closely resembles glanders and occurs chiefly in Burma, Malaya, Indo-China, Ceylon and South Africa. Rats are probably the chief natural hosts. The causal agent, *Malleomyces pseudomallei*, is closely related to *Malleomyces b. mallei*, and it produces a slowly developing infection in rats which can, during the course of the disease, act as active disseminators of the infection. A bacillus resembling *Malleomyces pseudomallei* has also been isolated from apparently healthy wild rats in the vicinity of Kuala Lumpur.

Salmonellosis, Escherichia coli and paracolon infections

Although numerous wild birds and mammals have been systematically examined for the presence of *Salmonellae*, comparatively few cases

have been recorded. Ever since the well-known work of Danysz (1900), who showed that enzootic disease in field mice was associated with a *Salmonella* (at that time called the Danysz bacillus, and which was probably a variety of *S. enteritidis* , natural outbreaks of *Salmonella* infection, especially due to *S. typhimurium* and *S. enteritidis* have been reported in rodents, with symptoms varying from fatal septicemia to simply a carriage state. In the United Kingdom, Savage and Read (1913), quoted by Savage and White (1923), found 5 of 41 rats positive bacteriologically, and Savage and White (1923) found *S. enteritidis* in 6 of a sample of 96 rats in the vicinity of two slaughterhouses. Khalil (1938) found several types of *Salmonella* in 7.3 percent of 750 Liverpool rats (70 percent brown and 30 percent black), i. e., *S. enteritidis*, *S. typhimurium*, *S. newport* and *S. thomson*, and Bruner and Moran (1949) in the United States pointed out that the following types could be recovered from rats and mice : *S. paratyphi* B., *S. anatum*, *S. newport*, *S. newington*, *S. montevideo*, *S. tennessee* and *S. salinates*. Workers in Mexico have also confirmed the existence of *S. pensacola* in rats (Varela, Olarte and Mata, 1948). Further information is provided by Wilführ and Wendtlandt (1921) and Spray (1926) to support the contention that food poisoning in man commonly arises through the contamination of food by infected rodents, and Hülphers and Henricson (1943) found 48 of 186 rats from various parts of Sweden to be carrying potentially dangerous *Salmonella*. *S. typhimurium* also appears to be associated with sea birds; Van Dorssen described a case in a seagull in 1935, and *S. paratyphi* B. has been isolated recently from the same source by Kumerloeve and Steiniger (1952). By the nature of their scavenging habits, it is to be expected that those birds would sooner or later come in contact with these organisms. A rhamnose-negative *S. typhimurium* was also isolated by Van Dorssen (1953) from pigeons. Magpies, some other birds and foxes have been identified in South Australia (Watts, 1951) as being responsible for spreading salmonellosis (*S. typhimurium*) in sheep stocks by contamination of drinking water, and in a dry country with little surface water this fact may be of considerable importance. Further evidence concerning salmonellosis in Australian wildlife is available in an article by Lee and MacKerras (1955). *S. typhimurium* has also been isolated from a sparrow by Bouvier *et al.* (1955) in Switzerland; they suggested the possibility that the bird had become infected from eating cultures put down to infect rats. *S. pullorum* has been recovered from a magpie in the United Kingdom (Jennings, 1954), and *S. gallinarum* has been found in rooks in the same country

by Harbourne (1955). In the latter case, the rooks acted as an important natural reservoir, and widespread outbreaks of the disease in domestic fowls in the area might well have been due to contamination from this source. It was the concensus of opinion in the area affected that other factors quite apart from the infection in poultry were obviously involved. *S. gallinarum* has also been isolated from a curlew in the United Kingdom (Jennings, 1954), and Ugorski (1952) has claimed that foxes in Europe can sometimes act as hosts for *S. derby*. In game, *S. pullorum* has in the past been considered an important disease in young partridges (Fitzgerald, 1946), but this contention has been based mainly on circumstantial evidence, and while it is quite conceivable that a limited number of young partridge chicks could be infected from carrier fosterhens, it is unlikely that this infection is of much importance in birds living wholly in the wild state. Bacteriological examination of many partridges by the author has failed to reveal the presence of this organism. From a liver culture obtained from an adult pheasant, Cass and Williams (1947) isolated *S. pullorum* and claimed this to be the first recorded case in this species in the United States.

The precise nature of ulcerative enteritis in quail in the United States has never been satisfactorily determined, but Graham (1936) described the isolation of a *Salmonella* species from immature quail. Kirkpatrick, Moses and Baldini (1952) suggested the use of antibiotics in the feed, and this might even prove to be of value in the field under certain circumstances.

In partridges, Edwards and Bruner (1941) recorded the isolation of *S. illinois* in the United States, but this appears to be the only instance of the isolation of this strain from this particular species.

In a general survey of *Salmonella* in wildlife, Buxton and Field (1949) described the isolation of *S. typhimurium* from mice and rats, *S. enteritidis* from hedgehogs, and *S. thomson* from the house sparrow.

S. typhimurium has also been isolated from captive muskrats in the United States by Armstrong (1942), who suggested that this disease may well occur in nature. Chitty and Southern (1954) again drew attention to the part played by rats and mice in the contamination of foodstuff by their feces containing *Salmonella* and also by culture sold as "poisons" for use in their control. In the latter instance, *S. typhimurium* and *S. enteritidis* have frequently been employed, and mice or rats infected with one or other of these varieties might well constitute a serious

hazard to public health. Recently, it was brought to the author's notice that cases of *S. enteritidis* infection in the human population had been traced to oatmeal, presumably contaminated by rodents, which had apparently been eaten raw by the individuals involved. Jones and Wright (1936) also recorded the contamination of food by mice carrying *S. typhimurium*.

Apart from the true *Salmonella*, the following cases are perhaps of some interest. *Escherichia coli* has been isolated from the alimentary tract of a wild sea lion by Oppenheimer and Kelly (1952) and from pyemic lesions in rats being examined for plague in the United Kingdom; paracolon organisms have been shown to be associated with a disease in partridges and wood pigeons, almost indistinguishable histologically from *S. pullorum* infection (McDiarmid, 1953). The role of the paracolon group has not been considered of much significance in the causation of disease in man or animals, but recently in the United Kingdom cases have been diagnosed in man (Kernohan, 1952; Block, Milzer and Kerdeman, 1949; Dornall, 1948; Cronk, 1952). The disease is also recognized in the human subject in other countries. Hinshaw and McNeil (1946 a) recorded severe mortality in turkeys due to this type of organism in the United States and suggested rattlesnakes as possible reservoirs (1946 b). These authors also isolated the same type of organism from Californian lizards.

In the United Kingdom, as has already been noted, pullorum disease (bacillary white diarrhea) caused by *S. pullorum* has been blamed for mortality among partridges, but it now seems highly probable, because of the close similarity macroscopically and microscopically between this disease and "paracolon" infection, that some confusion has arisen as to the precise nature of the disease in question. It seems worth while to emphasize that a correct diagnosis must always be based on a laboratory examination and not simply on the appearance of the affected bird.

This paracolon infection may be important in creating false positives to the blood agglutination test for pullorum disease in domestic poultry. From time to time, reactors occur in accredited poultry flocks which cannot be explained satisfactorily. These reactors do not develop clinical symptoms and usually fail to react when tested 30 days later. It may be that at least some of these indefinite reactions arise from previous contact with paracolon organisms derived from wild birds.

Plague (*Pasteurella pestis*)

Apart from the fact that mice were known to have died in the "Black Death" in Constantinople in the fourteenth century, wild animals were first suspected of being involved in the general epidemiology of plague in 1893, when it was observed at Canton in China that heavy mortality in the local rat population preceeded the appearance of human cases. However, it is now well recognized that plague is epizootic in rats and certain other wild rodents in many countries throughout the world, and it is perhaps true to say that this disease was probably one of the first conditions in which a clear-cut association was demonstrated between man and a free-living wild host. The infection is readily spread by rat fleas (*Xenopsylla cheopis*) from rat to rat and to any other susceptible host such as man. The organisms, following the ingestion of blood, actually multiply in the alimentary tract of the flea and the infection of the subsequent host occurs through regurgitation during the sucking of blood. In wild rats, the principal lesions at autopsy are enlarged lymphatic glands, especially the cervicals, because the area in which they are situated is the main feeding site for the fleas; pleural effusion and enlargement of the spleen may also be present and the spleen substance may show white spots; the liver is frequently granular in appearance; and congestion of most of the viscera is a common finding. Petechial hemorrhages may be noticed throughout the body. The symptoms have been described in considerable detail in the report of the Plague Commission (1907) and by McAlister and Brooks (1914). Some rats which yield cultures show no macroscopic evidence of plague (Williams and Kemmerer, 1923).

Because of the small number of organisms in smears of tissues from wild rats which have died of plague, and the difficulty of isolating a pure culture from such animals, the direct infection of suitable experimental animals such as white rats or guinea pigs is usually resorted to, in order to confirm a tentative diagnosis. It will be appreciated from the above description of the lesions of plague that, without a detailed bacteriological examination, other conditions can readily be confused with it. *Pasteurella pseudotuberculosis* can, on occasion, give rise to a very similar picture. Considerable confusion has arisen concerning the precise classification of organisms isolated from plague-like lesions in wild rats in East Anglia, England, which were extremely difficult to classify and which Petrie and McAlister (1911) eventually designated *Past. pseudotuberculosis*. Eastwood

and Griffith (1914) recovered strains which seemed to resemble *Bacillus monocytogenes*, and McAlister and Brooks (1914) found organisms of the *gaertner* group. They also mentioned that trypanosomiasis could be a further complicating factor. Within comparatively recent years, much information has been published on the occurrence of *Pasteurella pestis* in other feral hosts. Jellison (1939) drew attention to the possible role of predatory and scavenger birds in relation to the epidemiology of sylvatic plague. Meyer, Holdenried, Burroughs and Jawetz (1943) recorded still further information pertaining to plague in ground squirrels in California, originally described by McCoy and Wherry (1909) and by McCoy (1910, 1911). One of the interesting features of the disease in ground squirrels is the occurrence of subacute infections with indications that the strains are apparently less virulent than those in some other species. The spermophile is recognized as a source of plague in the southern part of the U.S.S.R. as well as field mice, and the tarbagan is responsible for outbreaks of disease in tarbagan hunters in Manchuria and Mongolia.

Holdenried and Morlan (1955) also recovered plague-infected fleas in northern New Mexico from rodents. In South Africa, gerbilles and multi-mammate mice can act as reservoir hosts (Mitchell, 1921) and Pirie (1929) and Mitchell (1930) described a disease syndrome in veldt rodents which was probably a genuine *Pasteurella* infection. Later, Davis (1953) drew attention to the epizootic cycle in gerbilles. Many types of desert rodents, as well as their own particular predators, such as the yellow mongoose and suricat, have now been involved as carriers of the disease.

Several other papers have also appeared concerning the reservoir hosts for plague, but those quoted will probably serve to indicate the widespread nature of the problem and the constant interest which is being taken in this matter, which is of considerable public health importance in many countries.

Other conditions associated with Pasteurella

In free-living birds, pasteurellosis has not been widely recorded. *Past. aviseptica* was isolated from puffins by Suarez and Ilazabal (1941) in South America, and later by Kaschula and Truter (1951) from seagulls on Dassen Island. The disease apparently occurs in wild ducks

(Quortrup *et al.*, 1946), the pintail and mallard being the species mainly affected. It is interesting to note, however, that the strains of the organisms isolated from these ducks were slightly different in character from typical *Past. aviseptica* and were eventually classified as variants. Cases have been recorded in the coot by Raggi and Stratton (1954) in the United States and in the partridge by Jennings (1954) in the United Kingdom, who mentions that the lesions in the partridges consisted of pneumonia and fibrinous pericarditis. The disease has also occurred in game birds in the United States, such as the pheasant (Hudson, 1944). From the purple grackle and the American robin, Bivins (1953, 1955) isolated strains which produced typical lesions in fowls, but apparently the organisms were not recovered from the experimental birds. Recently, the author recovered a strain of *Pasteurella aviseptica* from a swallow which had just arrived in England from Africa. From the nature of the lesions in the lung, there was no doubt that the infection had been progressing for some time and that this was a clear-cut example of how a foreign strain of organism could be introduced as a result of migration.

There seems little doubt, therefore, that species of wild birds can, on occasion, become infected with *Pasteurella* and could act as reservoirs of infection for domestic species.

Pasteurellosis in wild mammals is apparently rare, although Cowan (1951) mentions "hemorrhagic septicemia" as one of the principal causes of epizootic death in bighorn sheep in Canada. He points out that the *Pasteurella* may be present even in healthy sheep, and the precise reasons why the disease should suddenly appear are not yet fully understood. Death can even occur within 24 hours, with few symptoms apart from petechial hemorrhages throughout the body, although most cases die within two to seven days.

Pasteurellosis has been recorded in hares in Finland (Muroma, 1951) and in Switzerland (Bouvier *et al.*, 1953), and squirrels have also been shown to be affected in the latter country (Bouvier *et al.*, 1952). Hülphers and Henricson (1943) isolated numerous strains of *Pasteurella* from wild rats in Sweden.

Pseudotuberculosis (*Pasteurella pseudotuberculosis*)

Past. pseudotuberculosis has been isolated from a variety of mammals and birds in different countries. The infection appears to be quite common in Europe and numerous cases have been recorded in recent years,

FIGURE 6. — Pseudotuberculosis (*Pasteurella*); pyemic lesions in the liver, spleen and kidney of a brown hare.

especially in hares. Records of infection with *Past. pseudotuberculosis* in hares are available from Yugoslavia (Zibert, 1939); Germany (Olt, 1937); Finland (Muroma, 1951); Denmark (Christiansen, 1939); Switzerland (Bouvier *et al.*, 1954), where it was pointed out that the disease has been common in that country for many years, and Sweden (Thal, 1953), where it was shown that strains isolated from hares could be used for the preparation of a living vaccine which protected guinea pigs against disease produced by virulent *Pasteurella*. This tends to confirm the author's opinion that the organisms isolated from wild hares may, in many cases, be atypical and pathogenic for that particular species, but not necessarily so for experimental animals. By passage, however, it may be possible to enhance their virulence for other species. The disease

in the hare is generally a pyemia with foci scattered throughout the body and varying greatly in severity. In a typical case, small necrotic nodules are distributed in the principal viscera, the liver, spleen and kidneys being particularly affected (Figure 6). In other cases, a considerable amount of exudation may occur in the pleural, pericardial and peritoneal cavities, and the organs may appear congested without the occurrence of typical necrotic nodules. It is an interesting feature of the disease that on the Agricultural Research Council's field station in Berkshire, England, where it has been confirmed in the wild hare population, the small experimental animals, such as rabbits and guinea pigs, fed on green food from the estate or folded on new leys, have remained free from this infection. Apart from hares, pseudotuberculosis has not so far been confirmed as a serious disease of free-living mammals, although it has also been observed in the United Kingdom in the rabbit (Clapham, 1953) and the bat (unpublished observation) by the author. The alteration in nomenclature from *Past. pseudotuberculosis rodentium* to *Past. pseudotuberculosis* appears to be quite justifiable, as the disease is certainly of much greater importance in birds than in mammals. A useful review of the early history of *Past. pseudotuberculosis* is contained in an article by Beaudette (1940), in which he also described the isolation of the organism from a blackbird and claimed this to be the first avian case recorded in the United States. In Denmark, Christiansen (1949) mentions the occurrence of pseudotuberculosis in partridges, pheasants, wood pigeons, wrens and pied flycatchers. Since 1952, the author has encountered numerous cases in wild birds in the United Kingdom; the strains so far studied have been derived from such varied species as the wood pigeon, stock dove, magpie, tree sparrow and wren, with the majority of cases occurring in the first two species. In addition, Clapham (1953) has isolated the organisms from the lark, jackdaw and rook in the United Kingdom. Jennings (1954) has also reported the isolation of this organism from multiple caseo-necrotic lesions in a coot. It is interesting to note that Pullar (1932) drew attention to the association between the Australian magpie and pyemic hepatitis in sheep.

In birds, the disease follows a similar course to that in hares, and pyemia is common. In an affected tree sparrow examined by the author, there was extensive damage to the intestinal mucosa, causing almost complete closure of the lumen. A considerable amount of infection must have been discharged by this bird, and it can readily be understood how an enzootic of this disease can occur in a population of birds

such as pigeons, commonly feeding in large numbers. In a severe outbreak in Hampshire in the south of England, over 40 dead stock doves were picked up in one small area (Clapham, 1953).

Pseudotuberculosis is probably of increasing importance because of the potential risk to the human population. Only some few cases have been recorded in man in the past, but several are now being recognized, the disease involving mainly the alimentary tract and the associated lymphatic glands; the possibility of an ingestion infection from fresh green salad crops contaminated by infected free-living hosts cannot be ignored.

Pseudotuberculosis (*Corynebacterium*)

Practically all the authentic records of *Corynebacterium pyogenes* infection in free-living wild mammals are confined to deer in Germany or North America. Schoop (1940) described 12 cases in roe deer, 2 in fallow and 1 in a red deer in Germany. Lungs, liver and kidneys were the commonly affected viscera, with multiple abscess formation. A case of pseudotuberculosis caused by *Corynebacterium* species was encountered by Hammersland and Joneschild (1937) in the United States and again by Seghetti and McKenny (1941), and Cowan (1951) described a chronic caseous lymphadenitis which appeared sporadically among mule deer in western Canada, causing pyemic lesions throughout the body. The so-called diphtheria in wood pigeons mentioned by Scone (1927) in the United Kingdom may have been due to *Corynebacterium*, although it is more likely, in the light of present knowledge, that tuberculosis or perhaps trichomoniasis was the primary cause.

Brucellosis

Because of its importance to veterinary and public health, increasing interest is being shown in determining the extent of brucellosis in nature. In many countries, vaccination with Strain 19 vaccine against *Br. abortus* is now practiced, with a view to the eventual eradication of the disease. By this and other methods of control, the amount of *Br. abortus* infection in some countries has already been curtailed sufficiently to enable such a policy to be undertaken; in fact, in a few countries, brucellosis in

cattle is now eradicated. It is therefore of considerable interest to determine if natural reservoirs of infection do, in fact, exist, so that the risk of further infection can be assessed if vaccination should eventually be dispensed with. Most of the available information concerns *Br. abortus* and *Br. melitensis.* Little was known about the third type, *Br. suis*, in wild animals, until the infection in hares was proved by Bendtsen, Christiansen and Thomsen (1954), who regarded these infected animals as a possible source of the disease in swine. The most likely animals to harbor *Brucella* must, however, be those of the ungulate group, and these have been largely selected by various workers for special investigation, such as that described by Katz (1941), who also mentioned the importance of those animals as possible causes of "breakdown" in eradication schemes for this disease in domestic cattle. Lee and Turner (1937) investigated the use of tube and rapid plate-testing techniques for demonstrating the presence of *Brucella* agglutinins in the blood of elk in the United States, and subsequently Moore (1947) in Canada carried out a survey of buffalo and elk herds to determine the extent of *Br. abortus* infection. He examined 186 blood samples from elk of both sexes and all ages and found that all were negative to the agglutination test. However, in buffalo, the majority of which were males, 6 (16.2 percent) reacted positively, 5 (13.5 percent) showed doubtful reactions, and 26 (70.3 percent) were negative; 5 of the 6 positives were males. Other cases of the infection have been described in deer and hares in Germany, as mentioned by Witte (1941), and also in the same country in the testis of a roebuck by Schiel (1936). In this instance, sterility was noticed in the deer in the vicinity from which the animal was obtained.

A roebuck affected with arthritis and tendovaginitis was shown to be affected in Germany by Preun (1938), and Burgisser (1954) described the first case of brucellosis in the same species to be reported in Switzerland. The reason why most of the known cases in deer have been recorded in Germany, is probably because of the sporting interest shown in deer in that country. In chamois in Switzerland, Burgisser (1952) drew attention to the common findings of calcification of the lesions; all the animals he examined were from the same area and he suggested that transmission by coitus was not improbable. The strains which he recovered resembled *Br. abortus* but did not produce H_2S. Other isolated instances of susceptibility to *Brucella* infection have been recorded in buffaloes in the U.S.S.R. by Agababyan (1940), and reindeer in that country are also said to be susceptible.

In the United Kingdom, the author (1951) made an attempt to survey the wild deer population in the south of England for the presence of *Brucella* infection. A total of 80 deer of three different species was examined by the blood seroagglutination test. The species of the deer were fallow, Japanese and roe; the blood samples were collected as soon as the animals were killed by shooting, and the agglutination test was performed according to the standard technique used for cattle in the United Kingdom; 11 of the 80 samples showed demonstrable agglutinins — of these 5 reacted at dilutions of 1/40, 4 at 1/20 and 2 at 1/10. All were from fallow or Japanese deer. These findings suggest that *Brucella* infection does, in fact, occur in deer in the United Kingdom, and must therefore be considered as a potential hazard in any eventual eradication program. This view is supported by reports from gamekeepers and foresters who have seen aborted deer fetuses. Unfortunately, so far none has been obtained for bacteriological examination. In small wild rodents, Bosworth (1937) has described an isolated case of *Br. abortus* in the wild brown rat in the United Kingdom and brucellosis has been recorded in rats on the European continent by Karkadinovsky (1936) and again by Kolesnik (1941). The present author, however, failed by cultural and biological methods to demonstrate *Br. abortus* infection in 13 brown rats which had gained access to an isolation building in which cattle affected with brucellosis were housed. Before being killed, these rats had every opportunity to ingest portions of aborted placenta and the bedding was highly contaminated with discharges from the infected cows. It appears, therefore, that the rat is highly resistant to brucellosis. Moreover, Menton (1937) examined 200 rats caught in Staffordshire and failed to detect *Br. abortus* agglutinins in any of them.

Some evidence is also available concerning the disease in hares and rabbits. Selmi (1941) recorded the disease in rabbits in Italy and *Br. melitensis* infection has been confirmed in hares in France by Jacotot and Vallée (1951 a and b). Other records of brucellosis in rabbits have been given by Roux and Bouvier (1946) in Switzerland. Burgisser (1949, 1951) stated that as the strains of *Brucella* from Swiss hares differed from the ordinary three well-known types, they should be considered as a separate type, *Brucella leporis* (Figure 7). As previously mentioned, hares have also been shown to be affected with *Br. suis* in Denmark (Bendtsen *et al.*, 1954). In a recent publication, Thomsen (1959) stated that hare brucellosis is particularly widespread in Denmark, Germany, Czechoslovakia, Romania and Switzerland, and that the disease must be considered

FIGURE 7. — Brucellosis in a Swiss hare; testicular lesion.

to be a constant source of infection to swine. He also remarked that no investigations appear to have been made outside Europe. An interesting description of brucellosis in rodents is contained in an article by Manzullo (1935), who described the occurrence of *Br. melitensis* in free-living wild pampas cavies near La Plata in Argentina.

Another unusual variation of brucellosis infection has been mentioned in the chamois by Schmid and Klinger (1953). According to these authors, it has not been definitely proved that the causal agent is in fact *Brucella*, but a *Brucella*-like organism has been isolated from the lesions which consisted of keratoconjunctivitis; epizootics of the disease can apparently occur. Further information on this subject is provided by Bouvier *et al.* (1954) from Switzerland. Attention has recently been drawn to another unusual type of infection of sheep in New Zealand (Buddle and Boyes, 1953). This infection is caused by an organism now known as *Brucella ovis*. As yet, the original source of the infection has not been adequately determined, although in view of Manzullo's work already referred to, and the Danish experience, a reservoir host may yet be discovered in the wild native fauna of New Zealand.

Tularemia

In 1911, McCoy, while investigating plague in California rodents, found a " plague-like " disease in ground squirrels, and in the following year, in collaboration with Chapin, isolated the organism responsible for the condition. They named the organism *Bact. tularense*, from Tulare County in California, where the disease was prevalent. The precise classification of this organism has never been completely satisfactory and it has been placed in various groups by different workers. It does, however, appear to be more closely allied to the *Brucella* group than any other, and is now generally referred to as *Brucella tularensis*. Subsequently, Francis (1919) showed that " deerfly fever " in man was due to infection with *Br. tularense*, transmitted by the bite of *Chrysops discalis* from infected jack rabbits, and at about the same time, it was recognized that the snowshoe and cottontail rabbits were also a source of infection. Later, Francis (1923) recovered the organism from rabbits exposed for sale in Washington, and " rabbit fever " was recognized in the workers handling these rabbits. Infection has also been shown to be tick-transmitted, the tick in question being *Dermacentor andersoni* (Parker, Spencer and Francis, 1924). There is no doubt that much of the fundamental knowledge of tularemia was due to the work of Francis and his colleagues at the Washington Public Health Bureau. Subsequently, it was shown that wood mice could also be affected, and it was even considered that the fall in population of grouse in Montana might, in some way, be related to this disease. Further information on tularemia in the United States was furnished by reports describing its occurrence in beaver by Hammersland and Joneschild (1940) and by Scott (1940). The disease has since been encountered on an epizootic scale in beaver by Jellison *et al.* (1942), who showed that it might not be necessary for vectors to be present to spread the infection. They postulated the probability of direct infection from water, especially when stagnant, probably contaminated by some other animals, such as field mice, which were also proved infected in the enzootic areas. These workers quoted from the work of Seton (1929) to show that epizootics in beaver were well known many years ago, although not proved at that time to be associated with *Br. tularensis*. The muskrat, as well as the beaver, has been found infected in Canada by Langford (1954), and by Parker *et al.* (1951) in the United States. Kohls and Steinhaus (1943) also described the disease in shrews and field mice.

Tularemia has been confirmed in field rabbits in central Germany, and Bouvier *et al.* (1954) pointed out that the appearance of tularemia in France in 1945 could be the result of importation of hares from central Europe. The disease appeared in Belgium in 1950 and was probably introduced by field mice from France. The first case in hares in Switzerland was recorded in 1951 (Bouvier *et al.*), and this occurred practically on the French border. Water voles are considered as the principal source of infection in the U.S.S.R. (Roubakine, 1930; Voskresensky, 1943). Susliks have also been found infected in the U.S.S.R. by Tumansky and Kolesnikova (1935).

In the various species of wild animals so far found affected with tularemia, the lesions vary in appearance and sometimes are not unlike those of plague; consequently, this must always be considered carefully in differential diagnosis. It seems probable that much of the mortality leading to marked fluctuations in various rodent populations, i. e., the snowshoe rabbits in North America and the lemmings in Scandinavia, may be associated with *Br. tularensis*. The importance of the fluctuation in numbers of the food animals of the fur-bearing predators has been amply stressed by Elton and his co-workers at Oxford, who have over many years shown conclusively that mortality, especially in the snowshoe rabbit, plays a vital part in the fur-trading records of the Hudson Bay Company. It is, in fact, quite possible to predict good and bad trapping seasons, based mainly on the ten-year cycle of this species.

In Norway and Sweden, human infections have been traced to infected wild hares. Thjotta (1930, 1931 b) and Olin (1938) stressed the fact that human epidemics coincided with rodent epizootics. Thjotta (1931 a) drew attention to the "lemming fever" occurring in man as a result of drinking water contaminated by lemmings. In North America, Parker *et al.* (1951) mentioned the occurrence of over 100 cases of tularemia in the human subject, contracted from handling or skinning infected beaver or muskrats; most of these apparently occurred in the Northwestern States, but some were recorded in Canada and Alaska. These authors suggested that those probably represented only a fraction of the total cases, because of the mild nature of the disease in many subjects. Domestic animals, apart from sheep, do not appear to be readily infected.

The appearance of tularemia in water voles, muskrats and beaver leads to a serious consideration of the possibility of contamination of surface water supplies from, for example, the urine of affected animals.

In the U.S.S.R., Miller (1935) was probably the first to show the presence of *Br. tularensis* in a natural water supply, although previously in 1926 and 1928, serious epidemics in man had occurred in the U. S. S. R. In these, rat hunters were almost exclusively affected and it was found that water voles were the main source of infection. Further serious outbreaks of the disease in the human population were recorded by Karpoff and Antonoff (1936), Sinai and Voskresensky (1943) and Schmidt (1947), and appeared to be associated with water supplies contaminated by rodents. In the United States, Jellison *et al.* (1950) also drew attention to the risk of water-borne infection in human beings. It is interesting to note that concentrations of chlorine, such as are commonly used to treat "doubtful" water supplies, are sufficient to kill *Br. tularensis* (Foote *et al.*, 1943).

From the evidence now available, it is apparent that carriers of *Br. tularensis* abound in nature, particularly in rodents, in many countries throughout the world, especially in the American and European continents and in Japan, and that they constitute a very real hazard, directly and indirectly, to public health.

Botulism

Apart from botulism, few cases of clostridial infection or intoxications have been described in free-living wild animals; *Cl. botulinum* is, however, one of the important disease-producing agents in wildlife. There can be little doubt that botulism is a universal problem, and although so far almost entirely confined to wild fowl, especially in the United States, it constitutes a matter of considerable importance with its various implications to veterinary and public health. Since Wetmore (1918) described "duck sickness" in Utah, United States, numerous publications have appeared on this subject; only a representative sample of the available information can be discussed here. Gunnison and Coleman (1932) described outbreaks of intoxication by *Cl. botulinum* type C in Canada and the United States; Kalmbach and Gunderson (1934) and Kalmbach (1935) drew attention to the widespread nature of the infection, claiming it to be a potential world-wide hazard to wild fowl. Later, Kalmbach provided more useful information on this infection in wild fowl in the United States, Canada and Australia, and in 1939 he mentioned the occurrence of botulism in passerine birds, gulls, terns,

hawks, herons, grebes, cormorants and pelicans. There appeared to be little or no variation in species susceptibility. " Western duck sickness, " as this infection was named, received considerable attention, and suggestions for the control of botulism in wild fowl were made by Coburn and Quortrup (1939) at the Fourth North American Wildlife Conference.

In addition to wild ducks, botulism has been recorded in predators in the United States; i. e., the fox can apparently contract the infection (Pyle and Brown, 1939). Kalmbach (1939), however, pointed out that American vultures were resistant to experimental infection. He stressed the fact that this species must be exposed to considerable risk in nature and that the cause of its resistance merited further study. Other contributions have been made by Quortrup and Shillinger (1941), with an extensive list of species affected and details of a considerable number of post-mortem examinations. Later, Quortrup and Sudheimer (1943) showed that type C could grow well and elaborate toxin in the decaying vegetation of duck marshes.

Other clostridial infections

Apart from botulism, practically no evidence is available concerning authentic cases of clostridial infection in wild animals, although McKenney (1938) described a case of malignant edema in deer in the United States. Another observation was made by Taylor and Gordon (1940) when engaged in a study of grass sickness in horses in Scotland. While searching for possible etiological agents and studying the intestinal flora of various species, these workers demonstrated the presence of *Clostridium welchii*, type B, in the apparently normal intestine of a wild rabbit. They pointed out the importance of this finding in relation to the epizootiological aspect of lamb dysentery, as the wild rabbit might be a factor in the dissemination of this disease. Wild hares in the United Kingdom frequently die suddenly of a shock-like phenomenon when grazing on lush pastures, especially after frost. Although no bacteriological work has so far been done on this problem, the author has found small hemorrhagic zones in the stomachs of these animals. This lesion is very similar to that found in sheep dying from *Cl. septicum* infection and may represent the effect of this or a similar organism in the hare. " Shock disease " is, of course, a well-known entity in snowshoe rabbits in North America,

although perhaps of somewhat different etiology (Green and Larson, 1938). In view of the importance of sudden deaths, commonly considered to be due either to enterotoxemia or " bloat " in the domestic herbivora, further investigation of these cases in hares might well be justified, as it might help to throw some light on the phenomenon of sudden shock-like deaths in grazing animals.

Anthrax

An unusual case of birds acting as reservoirs of anthrax infection was demonstrated by Shrewsbury and Barson in 1952 during a bacteriological study of house sparrows in the United Kingdom. Anthrax spores, apparently in a latent state, were discovered in the crops of these birds. It is interesting to speculate on the origin of these spores and on their possible significance in the epidemiology and epizootiology of anthrax. The disease has been detected in zebras, black wildebeest, hartebeest and springbuck in South Africa (Thomas and Neitz, 1933), and is also known to occur in the hippopotamus and elephant. Human cases, resulting from the consumption of meat obtained from wild animals found dead, have also been recorded in Africa.

Actinomycosis and related diseases

Diseases in wild animals due to organisms of the *Actinomycetes* group are not common. With few exceptions, those which have been recorded have been mainly sporadic in nature. *Actinomyces bovis* has been associated with encephalitis in deer (Ryff, 1953); another classical case has been described in a roe deer by Bouvier *et al.* (1952). There has also been a description of an actinomycotic lesion in a roe buck's skull in Germany (Bosse, 1938). Cowan (1951), in Canada, recorded a variety of lesions such as the classical lumpy jaw, lung abscesses and foot rot, which he attributed to *Actinomyces*, in deer, moose, elk, caribou, mountain goat and bighorn sheep, the latter being most commonly affected.

Actinomyces muris has been described in numerous publications, usually associated with " rat bite fever " (Waterson and Wedgwood, 1953), and there can be little doubt that rats and other small rodents can act as carriers of this organism. More recently, the author, in con-

junction with Austwick (1954), encountered an organism of the aerobic *Actinomycetes* group which is associated with a pneumonic condition in moles. The significance of the presence of this organism in mole lungs is not fully understood, although it has been shown to be pathogenic for mice by various routes of inoculation, producing a chronic infection, with typical lung lesions. Bergey (1939) states that the actinomyces, numbering about 62 species, are common soil parasites, but that strains have also been isolated from a variety of animals. Their precise pathological significance is, however, difficult to determine. *Actinomyces asteroides* has recently been recovered by the author from the lung of a jackdaw. The lung showed a typical actinomycotic granuloma and the organism was isolated in pure culture. This organism has previously been associated with lesions resembling pseudotuberculosis in the lungs and pleura of man in Europe, Asia, Africa, the United States and, more recently, it has been recovered from the bovine udder in Australia (Munch-Petersen, 1954).

Listeriosis

Reports of listeriosis have so far been few in free-living wildlife and have been confined mainly to Scandinavia. Henricson (1943) recorded the disease in the hare, and Lillengen (1942) stated that capercaillie could also be affected. Vallée (1952) described the first recorded case in a hare in France, and Bouvier *et al.* (1954) discussed listeriosis in the same species in Switzerland. Recently the author has recovered *Listerella* from gray partridges in the vicinity of a flock of sheep also affected with the disease. Pirie (1927) described a plague-like disease in gerbilles in South Africa, which in some instances was associated with the "Tiger River" bacillus, known at that time as *Listerella hepatolytica*. This particular infection is apparently harmless to man.

Erysipelas (*Erysipelothrix rhusiopathiae*)

Erysipelothrix rhusiopathiae infection, varying in severity from the septicemic stage to a purely carriage state, has been described in a variety of free-living mammals and birds. Wayson (1927) gave an interesting description of an extensive outbreak in wild field mice in the United

States. In Sweden, Hülphers and Henricson (1943) recorded that 6.6 percent of 257 rats were infected, and Stiles (1944) recovered the organism from the brown rat in Colorado. A northern chipmunk in Canada was shown to be infected (Connell, 1954). Probably the first case to be found in a hare in Switzerland was reported in 1954 by Bouvier *et al.*, and Wellmann and Leibke (1960) have demonstrated the presence of bacilli and antibody in wild pigs in Europe.

Several species of birds have been found to be infected. Pheasants in Italy, although perhaps not truly in the wild state (Vianello, 1938), wild ducks in Germany (Bourgeois, 1944), the wood thrush (Lebeda, 1940) in the same country, and the white stork and herring gull in Denmark (Christiansen, 1949) are European records. Cases are rare in the United Kingdom, but the Scottish red grouse has yielded cultures (Taylor, 1954), and the wild wood pigeon has been shown by the author to harbor a very similar, if not identical, organism.

Necrobacillosis

Necrobacillosis has been recorded in the muskrat in the United States by Lord (1953), and no doubt with further investigation more cases could be discovered. It is a common infection in tick-infested hill lambs in Scotland, resulting in pyemic lesions from which *Fusiformis necrophorus* can be isolated, and deer frequenting these areas probably have every opportunity for contracting the infection. So far, no confirmed cases have been reported. Recently, " lumpy jaw " in kangaroos in Australia has been assumed to be caused by this organism.

Infectious anemia of wild rats

A large proportion of apparently healthy adult wild rats carry *Bartonella muris*, but this organism is of no significance as the causation of disease, unless the rats are caught and subjected to some laboratory procedure such as splenectomy, which appears to precipitate a severe illness, frequently resulting in the death of the animal.

Bartonella infection of moles

Graham-Smith (1905) demonstrated the presence of very small bacilli in the red blood cells of 10 percent of moles examined at Cambridge. Since then, similar organisms have been observed in the blood of a variety of small mammals. For a detailed review of these parasites, see Weinman (1944).

Leptospirosis (*Leptospira icterohaemorrhagiae*)

A considerable amount of interest has been taken in the carriage of *Leptospira* by rats and other wild animals, and it has been shown quite clearly by many workers in different countries that the rat, especially *Rattus norvegicus*, is the most common carrier of *Lepto. icterohaemorrhagiae*, and constitutes a potent source of infection for man and domestic animals. Some of the many references are as follows: Japan, Ido *et al.* (1917); Germany, Uhlenhuth and Zuelzer (1921); Canada, Cameron and Irwin (1929); U.S.S.R., Ssinjuschina (1929); Netherlands, Schuffner (1934); and the United Kingdom, Balfour (1922), Stevenson (1922), Middleton (1929), Buchanan (1927), and Broom and Gibson (1953).

The disease in rats appears to cause practically no systemic disturbance and rarely is jaundice observed. The spirochetes appear to localize principally in the kidneys (Figure 8), often for long periods and without causing ill effects. Consequently, the urine is the principal source of infection for other animals and man; hence the prevalence of the disease in miners and in workers in fish markets, who are constantly exposed to a considerable risk from urine-contaminated surroundings. The incidence in wild rats varies considerably with age, and may be up to 50 percent, according to the age of the animal from which samples were obtained; the older the rat the more likelihood of its being a carrier. Young rats appear to be relatively free from spirochetes.

It is interesting to note that black rats and house mice are rarely affected, although field mice can on occasion show a considerable degree of infection (Ido *et al.*, 1917; Buchanan, 1927; and Bessemans and Thiry, 1929).

Leptospirosis has also been recorded in a variety of other wild animals. The red fox in the United Kingdom has been shown to be susceptible

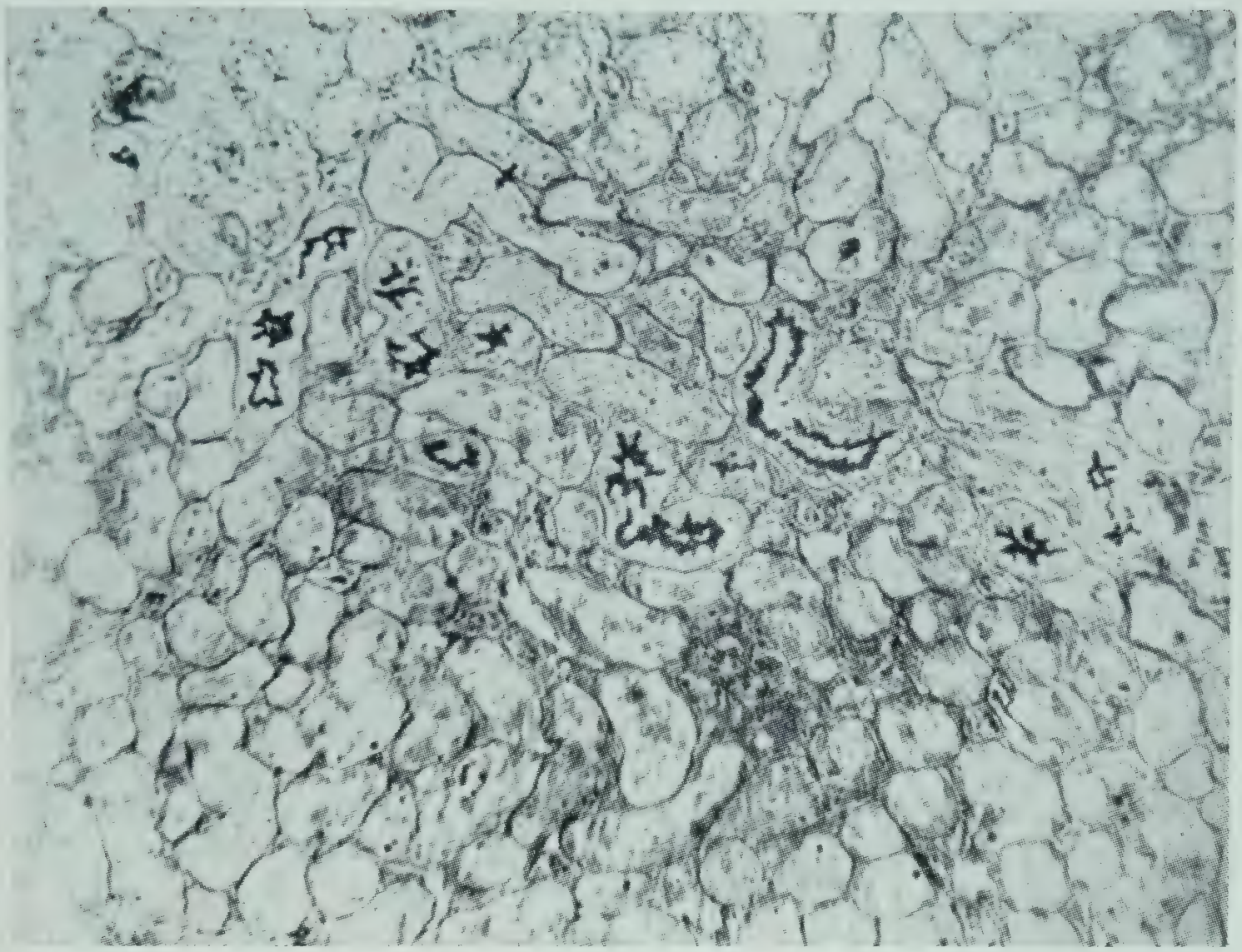


FIGURE 8. — Colonies of leptospires in the tubules of a rat's kidney (Levaditi).
Courtesy *Journal of Hygiene*

(Dunkin, 1926), and the author has personal evidence of a stoat carrying *Leptospirae* in the kidneys. These predators are frequently in contact with rats; in fact, rats constitute a considerable proportion of the foxes' diet, and it is therefore not surprising that this species of predator has been found to be affected. In country districts in the United Kingdom, wild foxes are commonly said to be affected with "yellows," a term which no doubt denotes leptospirosis, but the precise incidence of the disease is so far unknown.

Other infections with Leptospira spp.

A variety of other leptospires have been demonstrated in various species of wildlife. Babudieri (1953), in a most comprehensive review, lists a variety of leptospires and their predilection wild hosts in the

Italian rice fields. At least ten serological types have been described and at least eight species of rodents can act as reservoir hosts. He points out the interesting fact that *Lepto. bataviae* has as its main host a small species of mouse found only in rice fields, and human cases occur only in the same environment. The carriage rate in the mouse is about 25 percent according to Mino (1942). Further evidence regarding "swamp fever" in Poland is provided by Parnas and his co-workers (1961); small mammals with aquatic, field or domestic habitats are the main reservoirs of infection. The majority of strains isolated from wildlife have been *Lepto. grippotyphosa*, although five other varieties were also recorded. Van der Hoeden (1955) pointed out that jackals could act as an important natural reservoir of *Lepto. canicola*, the characteristic lesion in this species being interstitial nephritis, in many ways similar to that encountered in domestic dogs. *Lepto. grippotyphosa* has been identified in field mice and wood mice in the Danube valley and it appears probable that *Leptospira* may also be involved in a disease of raccoons in the United States. In this connection, it may be noted that raccoons dying of a distemper-like infection have shown jaundice, and leptospirosis has been suspected as a possible cause. Serological evidence of *Lepto. pomona* infection in deer has been available for some years in the United States, but recently experimental work has confirmed the susceptibility of deer to this infection and shown that abortion may occur in pregnant deer. Nevertheless, the clinical disease in nonpregnant deer is very mild and again there is evidence that deer may act as "silent" hosts for this organism (Trainer *et al.*, 1961). Feral house mice in Virginia have now been proved to carry *Lepto. ballum* in the endemic areas and an unidentified *Leptospira* has been isolated from an opossum. There is also a general article on the occurrence of spirochetes in wild fauna in the valleys of Muljab and Tedzen in Turkmenia by Latyshev and Pozgvaj (1936). In Japan, an autumnal disease, similar to a mild form of Weil's disease but which is never fatal, occurs in forestry and agricultural workers. This disease was first described by Ido, Ito and Wani (1917), and the natural reservoir for the causal agent (*Lepto. hebdomadis*) was shown to be field mice; 3 percent of the mice in the endemic areas were found to be carriers.

Vaccination against the infection is practiced in Japan in workers such as miners, who are constantly exposed to the risk of infection.

Lepto. bratislava has recently been isolated from a hedgehog in Scotland (Broom and Coghlan, 1960), and the same authors (1958) recov-

ered three other types, including *Lepto. ballum*, from rodents. The interesting feature of this work is the lack of antibody production by the wild hosts.

Rat bite fever

There can be little doubt that in the past there has been some confusion as to the exact etiology of rat bite fever in man, but present evidence indicates that at least two causal agents are involved, i. e., *Actinomyces muris* and *Spirillum minus* (Robertson, 1924). There is a widespread incidence of the disease in man, directly associated with exposure to rat bite; cases have been reported in the United Kingdom, Italy, Turkey, India, Dutch East Indies and the United States. Numerous strains of the spirillum have been isolated from rats and mice and all these strains appear to be similar (Schockaert, 1928). In Japan, Futaki *et al.* (1917) showed that 3 percent of the house rats carry the spirillum.

Relapsing fever

A variety of species of *Spirochaeta* have been shown to be associated with the disease syndrome known as relapsing fever in man.

In North Africa, the species of *Spirochaeta* associated with Moroccan relapsing fever has been isolated from ticks (*Ornithodorus marocarsus*) collected from the burrows of small wild mammals in the hill country, well away from the nearest human habitations, and this seems to indicate that these animals can act as a potential source of infection. Léger (1917) described *Spirochaeta crocidurae* isolated from a shrew mouse at Dakar and this organism is potentially pathogenic for man. Another species, *Spirochaeta normandi*, was recovered by Nicolle, Anderson and Colas-Belcour (1927) from ticks and subsequently from gerbilles in Tunis. Lingard (1907) also showed a spirillum in the bandicoot in India, which was probably the same species isolated by Carpano (1913) from the marmot. Another species, *Sp. didelphis*, has been recorded in the Brazilian oppossum by Vianna, Figueiredo and Cruz (1912).

In the ungulates in Africa, spirochetes have been noted in antelopes in Uganda by Bruce *et al.* (1911), and Todd and Wolback (1912) recorded the organisms in a roan antelope in Gambia. Wild pigs, porcupines and armadilloes have also been shown to be carriers.

Avian spirochetosis

Spirochetes have been demonstrated in the blood of certain wild birds, e. g., *Spirochaeta lagopodis* in the red grouse in the United Kingdom (Fantham, 1910), and similar organisms were isolated in culture from the little owl. Spirilla have also been demonstrated in the blood of snipe in Italy (Franchini, 1924). The precise significance of these findings is, however, difficult to assess and the possibility of post-mortem contamination must always be considered.

Rabbit syphilis

Rabbit syphilis is a naturally occurring disease of wild rabbits, caused by *Treponema cuniculi*. The condition was first described by Ross (1912) and the actual spirochetes were demonstrated by Bayon (1913).

The incidence varies greatly in wild rabbits in different countries and can, on occasion, reach 40 percent in the United Kingdom. The lesions on the genitalia and perineal region consist of small scaly patches with slight erosion, sometimes covered with a brownish crust. Occasionally, the nostrils and eyelids are also involved. Large numbers of spirochetes can be seen in scrapings from the affected areas.

Trep. cuniculi is indistinguishable, morphologically, from *Trep. pallidum*, but the two diseases caused by them are, in fact, quite distinct.

Further detailed information on this disease is contained in articles by Bessemans (1928), and by Bessemans and de Geest (1928).

2. DISEASES DUE TO FUNGI

Aspergillosis

Although mycosis is of common occurrence in birds maintained in zoological parks, records in free-living birds and mammals are comparatively rare. Davis and McClung (1940) described aspergillosis in herring gulls frequenting the mud flats in the vicinity of Boston harbor, and a similar case was recorded in the Thayer gull in Canada by Cowan (1943). Further information on the disease in seagulls was supplied by Herman and Rosen (1947), and it has also been observed in a puffin in Cornwall, England, by Motton (1945). During an investigation concerning the epidemiology of a peculiar virus infection, Surrey Dane (1948) mentioned the presence of *Aspergillus fumigatus* in the air sacs of manx shearwaters on the island of Skomer near the Welsh coast. The same disease has been reported in the black grouse, capercaillie, and a variety of wild fowl in Sweden, by Hülphers, Lillengen and Henricson (1941), in the mallard by Zeliff (1943), and in Hungarian partridges by Landy (1937). A single case has been recorded in a free-living Norwegian willow ptarmigan by Sörum (1950). Severe outbreaks in young wild pheasants in Berkshire, England, have been described by McDiarmid (1952), and Rothschild and Clay (1952) briefly drew attention to the occurrence of mycosis in wild wood pigeons. McDiarmid (1955) described 18 cases in birds in the United Kingdom, the species affected being the herring gull (Figure 9), jackdaw and wood pigeon (Figure 10). All were caused by *Aspergillus fumigatus*, with the exception of one wood pigeon from which *A. nidulans* was isolated, the first time that this species has been associated with disease in a wild bird. Aspergillosis has seldom been mentioned as the cause of disease in wild mammals, although it has occurred in the hare in Sweden (Hülphers *et al.*, 1941) and in the same species in the United Kingdom (author's observations). The author is

FIGURE 9. — Plaque-like lesions of aspergillosis in the abdominal viscera of a herring gull.

Courtesy *Journal of Comparative Pathology*

FIGURE 10. — Lesions of aspergillosis affecting the liver and pericardium of a wood pigeon.

Courtesy *Journal of Comparative Pathology*

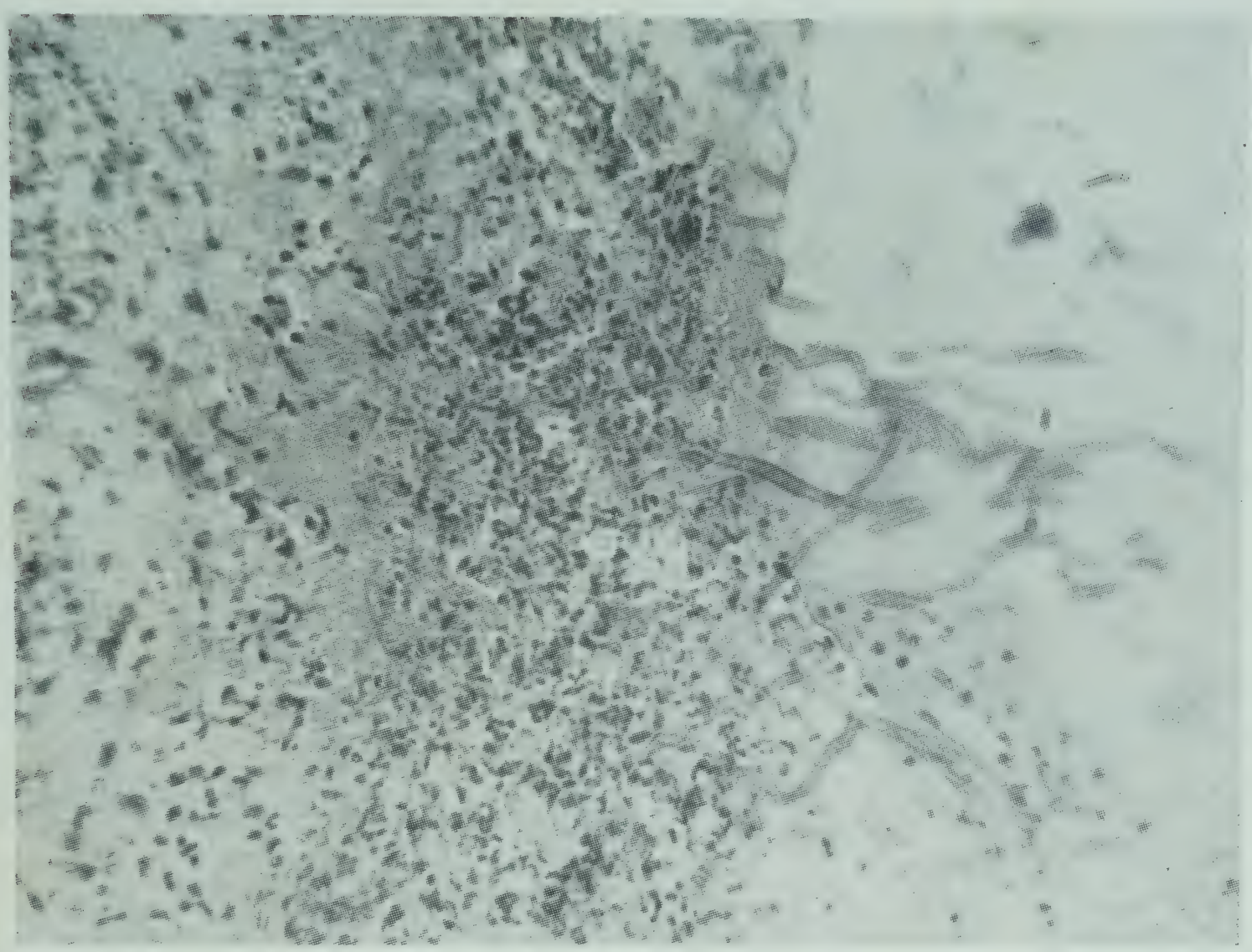


FIGURE 11. — Ramifications of aspergilli in the lung tissue of a juvenile pheasant (Hematoxylin and Eosin).

also aware of a suspected case in a weasel in England, but the precise nature of the fungus was not determined.

Irrespective of the host, the disease appears in most cases to produce the same macroscopic and microscopic lesions (Figure 11). Pulmonary air sac involvement is common, although in some instances the lesions may extend throughout the principal viscera, the kidneys frequently showing extensive involvement.

Economically, this disease is of considerable importance. Rearers of pheasants and other game birds can suffer severe losses if straw and weedseeds from combine harvesters are used for supplementing natural food reserves in covert (McDiarmid, 1952), and the view of the author is that aspergillosis has, undoubtedly, increased in wildlife in the United Kingdom since the introduction of highly mechanized farming. It is perhaps significant that the cases of aspergillosis confirmed in the United Kingdom and elsewhere have been confined mainly to water fowl or in

species particularly fond of grain. The former have every opportunity to acquire mycotic infection from decomposing vegetable matter in the vicinity of streams and ponds and on the seashore, especially after a period of low tides; the latter, no doubt, frequent heaps of fermenting weedseeds and damaged bales of combined straw. All the writer's cases of mycosis in Berkshire have been encountered since the advent of new farming methods in the area, and the scarcity of references to this disease in wildlife may in part be attributable to such changing conditions. The general mortality in wild birds, due to mycosis contracted in this way, may be considerable, although largely unnoticed. It is perhaps worthy of record that so far the author has never encountered a conjoint case of aspergillosis and tuberculosis in wildlife.

Coccidiodomycosis

Ashburn and Emmons (1942 a and b) in the United States first drew attention to the presence of a chronic granulomatous infection, frequently affecting the lungs of small wild rodents, associated with the presence of a fungus, *Coccidioides immitis*. Further reports appeared later (Emmons, 1943 a and b), and it is now clear that this infection, apart from the natural reservoirs in small wild rodents, can be of some importance in the causation of disease in domestic animals and man. This fungus was originally considered to be a protozoan, as the form which occurs in the tissues resembles an oocyst of a coccidium, and hence the generic name.

Although the disease is purely incidental in man, nevertheless, from the public health viewpoint, it is of considerable importance in the valleys of central and southern California, where the disease is endemic. The infection is probably contracted by inhalation of the chlamydospores in the dry, dusty season. It has been suggested that the organism is purely a soil saprophyte, but the work of Emmons has stressed the importance of animal reservoirs; the organism is certainly present in the soil but this could readily arise from fecal contamination by rodents. The early cases of the human disease arose near the San Joaquin river and the infection became well known as "San Joaquin valley" disease. Although originally recognized only in a chronic form, Dickson (1938) showed that another much more prevalent type was also present, namely "valley fever" or "desert fever," an influenza-like condition which

had not previously been associated with *Coccidioides*. There is probably a widespread incidence of this type in the human population in an endemic area, and the typical chronic cases constitute only a very small proportion of the total cases. Recently, the disease has been recognized in different species of domestic animals such as the cow, sheep and dog, and since cases have appeared in domestic stock out of the original area, e. g., in Arizona, Texas and Colorado, the reservoirs in the wild animal populations may be more widespread than has hitherto been believed. The species of small wild mammals already found to be infected in Arizona are pocket mice, kangaroo, rats and squirrels. These constitute the principal hosts and, according to Emmons, it may well be that the geographical distribution of the disease in human beings is determined largely by the presence of susceptible rodent reservoirs. It is worth pointing out that *Haplosporangium parvum* is closely related antigenically to this fungus and produces a very similar granuloma in a susceptible host. Indeed, many rodents, such as pocket mice, have been shown to harbor both agents simultaneously.

Haplomycosis

Haplomycosis is essentially a pulmonary disease of mammals caused by infection with a species of fungus known as *Haplosporangium*. This name was suggested by Emmons (1948) and the disease was first recognized in the United States by Emmons and Ashburn (1942) in the following species of wildlife: ground squirrel, pocket mouse, whitefooted mouse and kangaroo rat, in a semidesert area in Arizona. Dowding (1947 a and b, 1948) has reported infection in the whitefooted deer mouse and the red squirrel in Alberta and probably in the muskrat in British Columbia; Erickson (1949) demonstrated the infection in a beaver in Minnesota, and Jellison (1947, 1950) has also encountered the disease in the beaver, muskrat, pine squirrel, whitefooted mouse, rock rabbit, cottontail rabbit, mink, pine marten, skunk and weasel in Montana, in the whitefooted mouse in Idaho, and in wood rats in California. Carmichael (1951) noted that Emmons had also found the fungus in a pack rat in Texas. In eastern Kansas, both *Haplosporangium parvum* and *Histoplasma capsulatum* have been reported from raccoons, and the fungus has now also been isolated from the soil (Menges and Habermann, 1954). McDiarmid and Austwick (1954) found the disease to be

FIGURE 12. — Macroscopic lesions of adiospiromycosis on the surface of a mole's lung.

enzootic in moles in the United Kingdom. Large numbers of dead and dying moles were found on the Berkshire Downs during 1947 and again in 1953, and initial examinations showed evidence of pneumonia accompanied by granulomatous lesions (Figure 12). The lesions, varying greatly in size, were scattered throughout the lung substance, often protruding above the surface, and histological examination showed these reactions to be associated with the presence of a thick-walled spherule, 100 to 200 microns in diameter (Figure 13). These spherules were finally identified as *Haplosporangium parvum*. Many healthy moles were also found to be harboring the fungus in their lungs, and the exact significance of these bodies was further complicated by the presence in some specimens of at least one species of a pathogenic aerobic actinomycete (*Nocardia* sp.) and also a nematode which has not yet been classified.

The precise role of *Haplosporangium* in the periodic fluctuations in the mole population of the United Kingdom, which appear to occur about every six years (Elton, 1931), is not yet fully understood, but it appears probable that this fungus may to some extent be responsible for many of the deaths.

Jellison (1954) also recently demonstrated a *Haplosporangium* sp. in the lungs of rodents in Korea while searching for a possible etiological

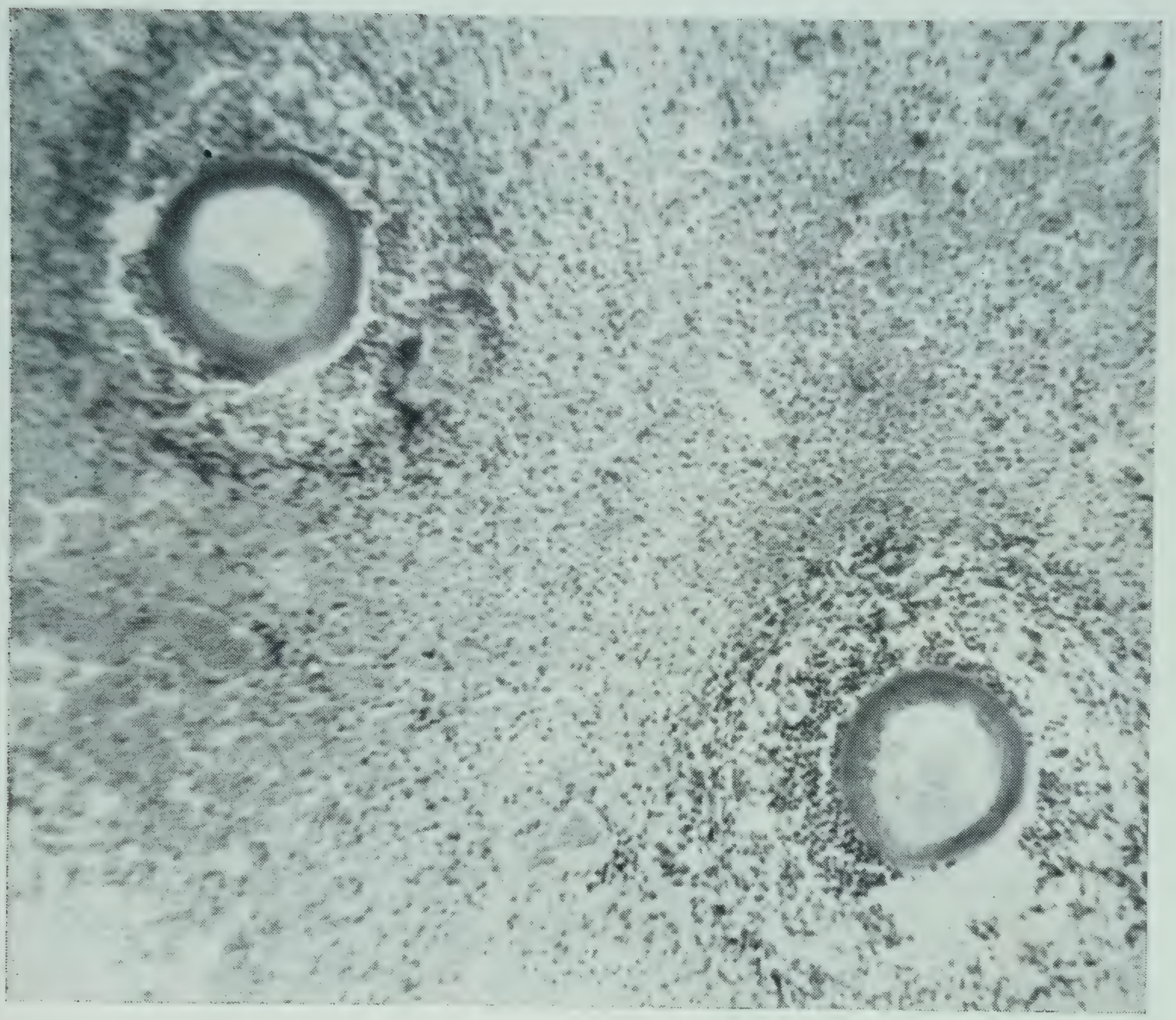


FIGURE 13. — Spherules of *Emmonsia* embedded in granulomatous lesions in a mole's lung.

factor in connection with "hemorrhagic fever," a serious disease of combatant troops in that area. It was originally suspected that the rodent population was the reservoir for the causal agent of this disease and Jellison examined 2,103 specimens of rodents during his survey; cysts of the fungus were found in 53 animals. Infection varied from one to hundreds of cysts. The animals as classified by Jellison were: "*Apodemus agrarius*, *A. peninsulae*, *Clethrionomys rufocanus*., *Orioetulus triton*, *Rattus* sp., and a single shrew of undetermined genus." All the rodents came from areas presumably endemic for "hemorrhagic fever."

This fungus may or may not be *H. parvum*; Jellison pointed out that although *H. parvum*, the only known pathogenic species of the genus,

had been found in many kinds of animals, it had not been reported from man and had not been encountered previously in Europe or Asia. It is therefore of interest that the isolates made by McDiarmid and Austwick provide, in addition to a new host, the first record of the disease in Europe. It is the author's opinion that the disease probably occurs in man, especially where an occupational risk is involved, such as in certain agricultural workers. At the time of writing, further information is being revealed concerning haplosporangiosis. Jellison and his co-workers have now demonstrated the presence of the fungus in the lungs of rodents collected in several other European countries, including Norway, Sweden and Finland (Jellison, Helminen and Vinson, 1960). It is suggested that the causal agent should now be called *Emmonsia*, and the disease it produces adiospiromycosis. Evidence is also accumulating to show that two main types exist: *Emmonsia parva* and *Emmonsia crescens* — distinguished largely by their size (Emmons and Jellison, 1960).

Histoplasmosis

Histoplasmosis is of ever-increasing importance in veterinary and public health (1952) and probably many reservoir hosts exist in nature. Emmons *et al.* (1947) and Emmons and Ashburn (1948) have drawn attention to rats and mice as possible sources of infection in the United States, and Menges *et al.* (1954) have also stressed the importance of raccoons and skunks. So far, however, there is no evidence of direct transmission from wild reservoirs to domestic stock or man (1952).

A mycotic disease of hares

At widely spaced intervals, about every six to seven years, hares have been found dead or dying from a variety of infections on the Berkshire Downs in England. In a proportion of these hares, yeast-like bodies have been present in the liver but, despite numerous attempts, no culture of the organism has been obtained. No other infection was found. The significance of these preliminary findings is, therefore, not yet fully understood.

Avian moniliasis

Thrush-like lesions varying in severity are sometimes seen in wild birds as well as in man, and they can on occasion cause serious mortality; pigeons, pheasants, partridges and grouse are the commonest species involved. Lesions may be found throughout the alimentary tract and recently the author has encountered severe enteritis caused by *Candida albicans* in jackdaws.

Sporotrichosis

Although seldom encountered in wild animals, rats are considered to contract infection with *Sporotrichum schencki* naturally (Hagan, 1943), and as there appears to be a higher incidence of this disease in agricultural workers than in those engaged in nonagricultural activities (Ainsworth, 1954), it may be possible that rodents play some part in the epidemiology of this disease.

Ringworm (dermatophytes)

With few exceptions, practically all the authentic references to ringworm in wildlife have only become available within the last 30 years and the cases described have been confined mainly to mammals. One of the earliest records was provided by Eddoes (1898), who mentioned the occurrence of ringworm in man contracted from a hedgehog in the United Kingdom. Unfortunately, the actual species of fungus was not determined. It was not until about 30 years later that further cases were recorded in wild gray mice in France by Du Bois (1929), and in this instance the causal agent was *Tricophyton granulosum.* He also discussed the general epizootiology of the condition. Connor (1932) in Australia and Shaw and Wampler (1933) in the United States then described favus, the so-called *Achorion quinckeanum* (*Trichophyton quinckeanum*) in mice and also in man, and further information on this subject was provided by Cook and Graham (1936) from the United States and by Poland (1938) from Europe. Watkins, in a discussion on a paper by Ainsworth (1954) on dermatophytes, mentioned the occurrence of an outbreak of favus in feral house mice, the species in this instance again being *Tricho-*

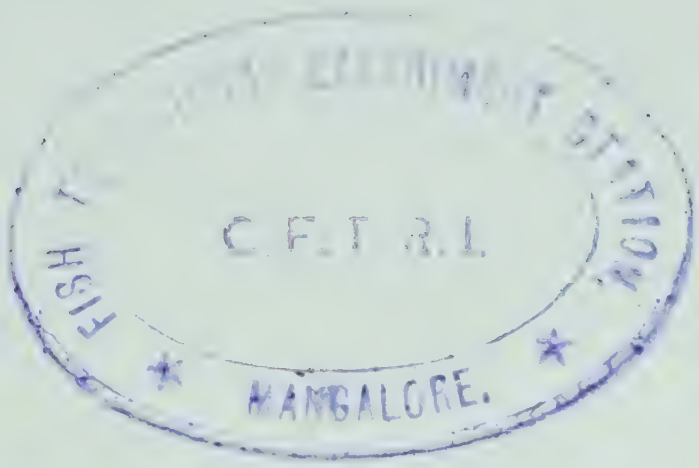
FIGURE 14. — Ringworm affecting the head of a black grouse.

phyton quinckeanum. In Japan, Hasegawa and Yamamoto (1936) described a ringworn infection of monkeys caused by *Microsporum fulvum*. The squirrel was cited as a new host for ringworm (*Trichophyton mentagrophytes*) by Delamater (1939), and later Blanc and Catanei (1944) encountered further cases in Moroccan squirrels. A ringworm infection of muskrats, apparently communicable to man, was described by Charles (1940) in the United States but again, unfortunately, the actual species of dermatophyte was not determined. Vilanova and Casanovase (1951) have also made clinical and mycological observations on an epidemic of *T. mentagrophytes* infection in rabbits, and Mantovani and Ceretto (1953) encountered ringworm in hares in the Piedmont region of Italy.

In birds, Pätiälä and Härö (1950) mention the occurrence of *Achorion gallinae* (*Trichophyton gallinae*) in blackgame in Finland (Figure 14).

There can be little doubt that this field of research is so far virtually untouched and would be well worthy of further investigation.

3. DISEASES DUE TO VIRUSES



Foot-and-mouth disease

In an important disease such as foot-and-mouth disease, it would perhaps have been expected that a considerable amount of interest would have been taken in the possibility of wild animals acting as reservoirs of virus and that this would have assumed even greater importance in a country such as the United Kingdom, depending essentially on a slaughter policy and eradication as opposed to vaccination for the control of the disease. It is surprising, therefore, that little appears to have been done to investigate this problem in wildlife, and much of the available information is based on individual case records rather than on concerted efforts, such as surveys of a variety of species as potential carriers. The only formal survey which has been made was reported by Elton (1937) working in the foot-and-mouth disease areas in the United Kingdom, and this survey was carried out essentially as an investigation of the varieties of wild animals present and mortality in those populations.

Longley (1937), in addition to cases in wild brown rats did, however, describe a naturally occurring infection in the hedgehog in the United Kingdom and McLauchlan and Henderson (1947) suggested that hedgehogs might sometimes be responsible for the spread of the disease. Other workers, especially in Germany, have recorded cases in deer. Thus Bartels and Claassen (1936 a and b) reported on the role of wild game in Germany in the epizootiology of the disease and further information on the part played by roe deer and red deer was furnished by Cohrs and Weber-Springe (1939) and by Sallinger (1939). The whole question of the importance of transmission by the *Cervidae* has now been considered in some detail on the European continent, and there seems to be little doubt that deer may, on occasion, constitute an important natural reservoir of virus. Outbreaks of the disease in domestic stock

in Rhodesia are attributed to disease in game, and the disease has been confirmed in kudu, eland, buffalo, warthog and bushpig. It is considered that the presence of the disease in wild game in Africa is a potent factor in the dissemination of the infection.

Foot-and-mouth disease has been described in elk in Sweden (Magnusson, 1939), and also in wild fauna in Denmark (Christiansen, 1939). The armadillo in South America (Campion, 1950) and the porcupine in Pakistan (Haq, 1951) have been shown to be susceptible to the inoculation of virus, and they may be of some importance in the spread of the disease, if they contract it naturally. An interesting speculation in the consideration of the method of spread of foot-and-mouth disease by wildlife is the part played by migratory birds, and Wilson and Matheson (1952) have discussed the part that the streams of migrants visiting England, particularly starlings, may play in the initiation of outbreaks. This possibility had already been suggested on previous occasions by Mettam (1914) and later by Bullough (1942). The points of landfall on the southeastern coast of England of migrants from the European continent coincide with the areas in which the initial cases of foot-and-mouth disease occur, and the authors rightly point out the very close connection between these vast flocks of birds and grazing animals, such as cattle or sheep. The risk of infection appears to be particularly great in trough-fed animals, as starlings congregate in large numbers at these feeding places.

If, in fact, birds are responsible for carrying foot-and-mouth disease virus, they probably do so purely passively, as experimental work with starlings, reported by Eccles (1939), has shown that the virus did not establish itself in the tissues of the bird. He also showed that starlings infected *per os* could excrete the virus for 10 to 26 hours in their droppings and that virus was still present 91 hours after external contamination of their feathers. It is therefore theoretically possible that birds ingesting, or coming in contact with virus, could under certain conditions remain carriers for sufficient time to transport the virus to England from some parts of the continent of Europe.

The effect of weather conditions has, however, not been determined and must always be considered in assessing the possible practical significance of this work. It should also be noted that Eccles (1939) examined 359 dead and 54 live birds from foot-and-mouth disease-infected farms in England during 1937-38 and failed to demonstrate virus in any of them.

Vesicular stomatitis

Evidence of antibody in deer and raccoons has now been obtained in Georgia and neighboring states of the United States by Karstad and his co-workers (1956), and experimental work has shown that this disease is of a mild type in deer (Karstad and Hanson, 1957).

Rift valley fever

Rift valley fever, which causes an epizootic hepatitis of cattle and sheep in the Rift valley, Kenya, and which will on occasion affect man, is caused by a virus which has been isolated from six different species of the mosquito eretmapodites in the Semliki forest in an uninhabited area in western Uganda by Smithburn, Haddow and Gillett (1948), and Smithburn, Haddow and Lumsden (1949) succeeded in transmitting the virus from mouse to lamb, from lamb to lamb, and from lamb to mouse, by the bites of the *E. chrysogaster* group. It seems probable that there is a sylvan cycle involving free-living animals, although evidence is lacking concerning the isolation of virus from free-living mammals or birds. Monkeys and certain species of rodents have now been shown to be experimentally susceptible (Rivers, 1952); rodents may, therefore, play a role in the ecology of this disease.

Rabies

Information accumulated over a period of years from many countries has shown conclusively that wild animals are often important in the transmission of rabies, especially in remote areas, far removed from human habitation. This has been proved in South Africa, where the yellow mongoose has been found infected. The jackal is considered to play a very important part in rabies epizootics in Southern Rhodesia, the northern Transvaal and South West Africa, and special measures of destruction are used against this species in control campaigns. In addition to jackals, the disease has been confirmed in ratels, civet cats, badgers, baboons and hyenas in Southern Rhodesia. The hyena has also been found infected in Northern Rhodesia. Evidence is available from India concerning the natural disease in tigers (Burton, 1950; Pan-

dit, 1951), and skunks are of considerable importance in the spread of the disease in certain parts of the United States. Ground squirrels, ordinary squirrels, foxes, wild cats, coyotes and wolves can all be involved, and it is well recognized that in Trinidad and other countries vampire and fruit-eating bats can act as symptomless carriers and transmit the disease to animals and man (Pawan, 1936, 1948).

In recent years, much interest has been taken in the feral hosts for rabies in Germany and considerable infection has been encountered in foxes (Schoop, 1950). The disease has also been recorded in the badger. In Yugoslavia, in 1950, wolves were the principal wild hosts and 47 cases in human beings were attributed to them; they are considered by Kodrnja (1952) to play an important part in the epidemiology of the disease. Chitty (1950) quotes apparent cases in the wildlife inquiry in Canada, and Cowan (1949) in the same country suggests that rabies is one of the several natural controls on the population of the arctic *Canidae*. In 1954, Plummer reported that "the most extensive enzootic of rabies ever known in Canada is in existence at the present time. It involves the Northwest Territories, practically all the province of Alberta, and certain northern districts of British Columbia, Saskatchewan, Manitoba and Quebec." He mentioned infection in foxes, wolves, coyotes, lynx, bear, rabbit, mouse, beaver, weasel, caribou and moose, and that attempts at control and eradication were based mainly on reduction of the susceptible wildlife population and the control of dogs. He considered that the fox is the most dangerous "travelling reservoir" because it often fails to kill its selected prey, thus passing on the virus. In Algeria, Herrenberger (1952) failed to isolate the virus from wild rats, which were considered to be a potential serious persistent reservoir of infection if they harbored rabies in an occult form.

One of the most important factors to be considered in the control of rabies in some countries is the migration or nonmigration of susceptible wildlife hosts, and little is known about this aspect of animal ecology in the enzootic areas. Some workers go so far as to suggest that in the United States rabies has been long established (since 1920) in the coyotes of the Western States, especially in California, and it is interesting to note that in 1922-32 many were shown to be infected. Cases have also been mentioned in ground squirrels, polecats, spotted genet and wildcats. Further information is available from the publications of Pritchett (1938) of the infection in gray squirrels; of Hirleman (1942) in foxes in Georgia; and of Tice and Evans (1942) in a fox in New York

State. Young (1953) discussed in general the epizootiology of rabies in Texas wildlife during 1946-51 and gave details of general control measures; in Colorado, it is suggested that rabies has existed there for over 100 years, especially in wolves. Also in 1954, Pullar and McIntosh gave a general description of the disease in wildlife in different countries, and Fagan (1953) discussed the difficult problems involved when free-living species act as reservoirs of the virus. Within the last few years, bats have caused grave concern in their obviously important role of rabies in the United States, especially in Florida and Pennsylvania, and Courter (1954) mentioned a rabid insectivorous bat attacking a human being; in California virus was recovered from a similar bat by Enright *et al.* (1955). The possibility of migrating bats carrying virus into Canada has also been stressed recently (1954).

At one time paralytic rabies was apparently widespread in Brazil, where infection is carried by the vampire bat, and Monila (1938) spoke of the disease reaching epizootic proportions on cattle ranches.

From this considerable amount of evidence, it appears that the domestic dog is not necessarily the most important reservoir of rabies, although by its close association with man, especially in urban areas, it must undoubtedly be involved directly in many human cases. The general impression is that the disease is widespread and probably enzootic in nature in certain countries, and a limited control program, based entirely on domestic animals, is likely to fail if the free-living fauna are not also taken into consideration and suitable trapping and poisoning programs instituted in order to diminish the number of predators in the affected area. A detailed description of rabies control measures in wildlife in Alberta is given by Ballantyne and O'Donoghue (1954), and these techniques could well be utilized elsewhere if the need arose.

Pseudorabies (Aujeszky's disease)

Information on pseudorabies in wildlife is meager, but Shope (1934, 1935) showed that brown rats could readily develop this disease in the midwestern region of the United States, and he also postulated that they could act as transmitting agents from farm to farm.

Psittacosis

Psittacosis is primarily an infection of birds, especially of the parrot family, caused by a group of closely related viruses, although within about the last 20 years it has been shown to occur also in other species, such as pigeons (Pinkerton and Swank, 1940). Frequently, the term "ornithosis" has been used to describe the infections with strains of virus slightly different from the classical type.

Haagen and Mauer (1938) readily detected virus from juvenile fulmar petrels taken on the Faeroe Islands. The birds were first suspected as reservoir hosts by Rasmussen (1938), following epidemics of atypical pneumonia in the human population on the island, affecting especially the women. It was thought that probably the human infection was due to inhalation of virus during the process of plucking the young birds.

The significance of the presence of psittacosis virus in sea birds such as fulmar petrels was discussed in 1939 in the *Lancet*, and Miles and Shrivastav (1951), who described the initial outbreak in fulmars in 1933, showed that herring gulls and lesser black-backed gulls could also be affected, and provided evidence to indicate that these infections were probably long established in the gulls before the fulmars became infected. Mykytowycz, Surrey Dane and Beech (1955) have recently described ornithosis in Australian petrels.

Java sparrows and thrushes can also apparently contract this disease, and a serious outbreak in 1929-30 was finally traced to the importation of South American green Amazon parrots. Fortunately, it appears that the virus derived from domestic pigeons is not so virulent for man as are strains from psittacine birds. In Australia, psittacosis is enzootic in wild parrots (Burnet, 1935), and antibody can be demonstrated in many other wild species. Rubin *et al.* (1951) drew attention to the occurrence of psittacosis-like infection in wild snowy egrets in south-eastern Louisiana and compared the close association of this condition with human pneumonitis (Borg strain). Subsequently, Rubin (1954) described the same infection in captive egrets and suggested that one or more of these birds were probably infected prior to capture.

It appears from all the foregoing evidence that psittacosis is essentially a natural disease of free-living wild species of birds and that cases detected in man are purely incidental. A high occupational risk is involved in certain communities, especially in Faeroe Islanders, in handling

large numbers of immature fulmar petrels and also by those people brought in direct contact with psittacine birds.

Opossum disease

A very closely related virus appeared to be responsible for deaths in opossum shortly after capture in Colombia (Roca-Garcia, 1949), and opossum disease may well occur under natural conditions.

Pox diseases in birds

Pox-like diseases occur in many wild birds and it has been frequently suggested that the infections now met with in different species probably arose originally from variants of a single virus, subsequently adapted to different host species. According to Holmes (1948), however, there are at least four strains of the virus which he has named *Borreliota avium*. It is interesting, therefore, to find that French and Reeves (1954), studying pox viruses from naturally infected mosquitos in Victoria and New South Wales, have succeeded in growing different varieties on chick embryo and have shown that five of these viruses may well have been acquired from wild birds in the area.

Pox was first observed in sparrows by Shattock (1898), and subsequently McGaughey and Burnet (1945) described the disease in the same species in Wales; the principal lesions appeared to be dry, black crusts in the vicinity of the mouth. The disease could apparently prove fatal to sparrows and canaries and in this way resembled canary pox.

The susceptibility of magpies to pigeon pox was mentioned by Geurdon (1941), and further records of pox-like lesions in free-living birds are recorded in the mourning dove in Illinois by Kossack and Hanson (1954), who stressed that it is a potential cause of mortality in this species. Syverton and Cowan (1944) found the disease also in the sooty grouse, on Vancouver Island, and quail and partridges in the free-living state are known to acquire this disease. A severe outbreak of pox in pheasants, maintained under semiwild conditions, was described by Dobson (1937) in the United Kingdom, and the author has frequently seen the disease in wild pigeons. Suspensions of dried crusts from a naturally occurring case will readily cause the production of typical lesions if applied to a scarified area on a domestic pigeon. The author's belief is

that considerable confusion has occurred in the past concerning true pigeon pox and trichomoniasis in wild pigeons in the United Kingdom and elsewhere.

Contagious ecthyma of sheep

The only record of contagious ecthyma of sheep in free-living wildlife concerns Rocky Mountain bighorn sheep in Canada (Conell, 1954), and it is possible that they may have contracted the infection from domestic sheep in the vicinity.

Fowl plague (true fowl pest)

Wild birds have been said to suffer from fowl plague, such as the pheasant (Hagan, 1943), but so far the present evidence is not sufficiently convincing to consider this infection as an important disease entity in wildlife.

Puffinosis

For many years, a disease of manx shearwaters has been recognized on the islands of Skokholm and Skomer near the Welsh coast of the United Kingdom. Herring gulls also appeared to be affected; varied symptoms were noted in the shearwaters, such as conjunctivitis and spastic extension of the legs, in addition to blisters on the feet (Figure 15). Surrey Dane (1948) and Surrey Dane *et al.* (1953) described the condition in considerable detail and mentioned, in particular, the peculiar lesions on the webs of the feet. This disease was further investigated by Miles and Stoker (1948), who referred to it as an epizootic caused by a virus which was filterable and could be cultivated on the chorioallantoic membrane of the developing chick embryo. It is apparently also infective for ducks and pigeons (Stoker and Miles, 1953) and has even been isolated from mites collected from the shearwaters (Surrey Dane *et al.*, 1953). The most interesting features of the disease are the high fatality rate in the juvenile birds, the method of infection, which appears to be mainly direct contact with an infected surface, i. e., stones, etc., contaminated by other shearwaters, and the characteristic foot lesions. Small consolidated areas may also be observed in the lungs in some specimens.

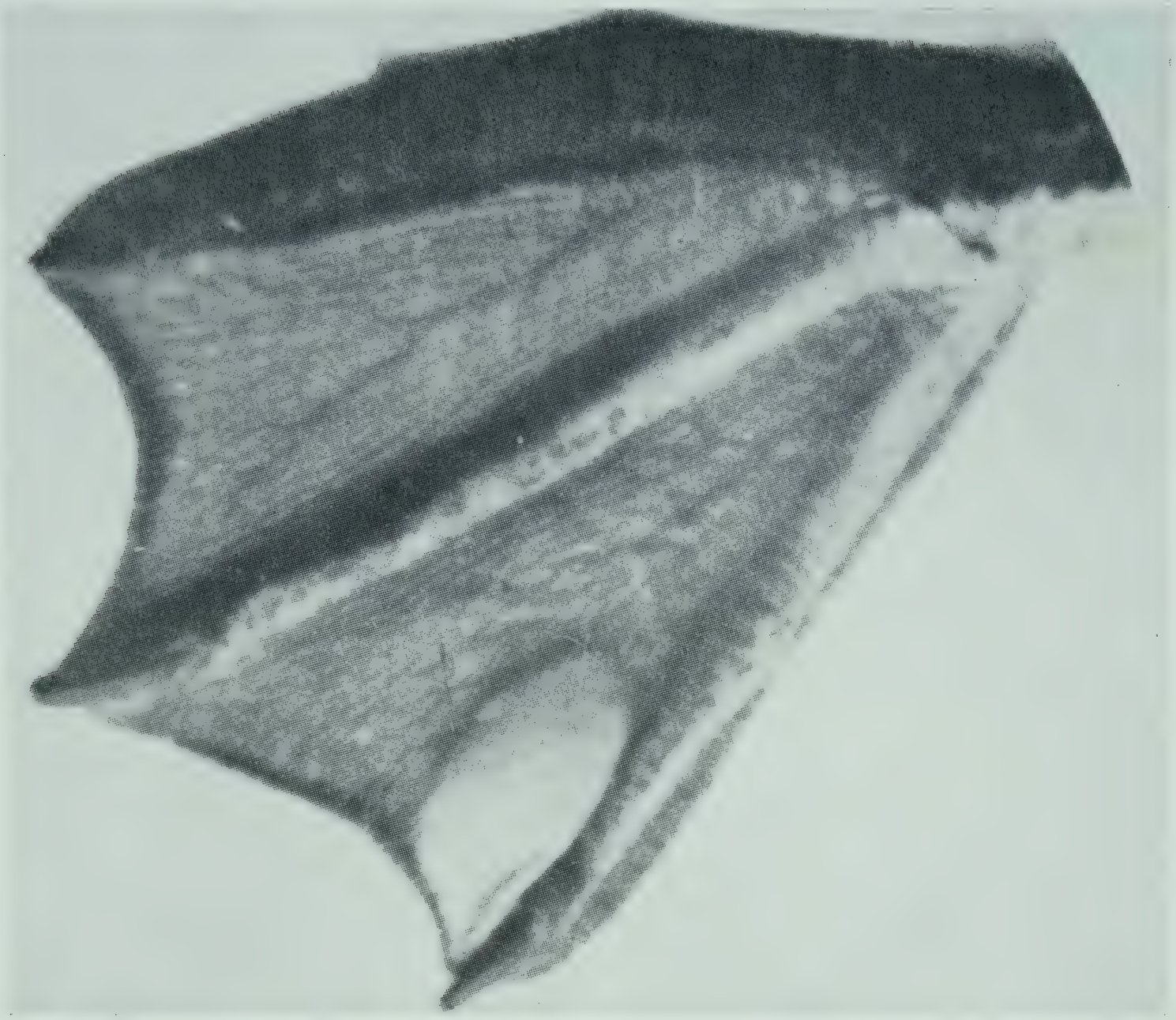


FIGURE 15. — Foot lesions in a Manx shearwater caused by the virus of puffinosis. Courtesy *Journal of Animal Ecology*

Newcastle disease (pseudo-fowl pest): (also referred to in the United Kingdom as fowl pest)

In several aspects, Newcastle disease is very similar to fowl plague and several reports of wild birds acting as carriers of the causal virus are now available from different countries. The virus was recognized in a free-living juvenile starling by Gillespie, Kessel and Fabricant (1950) in the United States, and subsequently the disease was confirmed in English sparrows in the same country by Gustafson and Moses (1953 a). These authors also gave a good general summary of existing knowledge about the infection in wild birds (1953 b). In the United Kingdom, the first case in wildlife was recorded by Wilson (1950), who isolated the virus from a gannet from the Orkney Islands near the Scottish coast.

Shortly afterwards, Blaxland (1951) demonstrated infection in shags and cormorants in the Hebrides. It was presumed that these birds had probably acquired infection, directly or indirectly, from fowl offal discarded from passing ships. From India, there is evidence that 35 partridges died in an outbreak of the disease (Parnaik and Dixit, 1953). In Germany, Zuydam (1951), after the examination of rats taken on an infected farm, suggested that these animals were of little importance in the spread of the infection. Popovič (1951) considered pigeons as possible spreaders of infection in Belgrade, and Mantovani and Ceretto (1953) encountered the disease in pheasants and partridges in the Piedmont area in Italy. African gray parrots in Kenya are also known to be subject to this disease.

Other virus infections in birds

Among other conditions in wild birds caused by viruses may be considered avian infectious laryngo-tracheitis, which apparently sometimes occurs in pheasants, and epizootic bronchitis, which has been said to occur in quail (Olson, 1950) in the United States. Mortality in blackbirds and mistlethrushes in northern Italy has also been said by Todd (1930) to be associated with a virus infection, in many ways resembling fowl plague, although the fowl was apparently not susceptible to this new virus.

Infectious sinusitis

In the United Kingdom during 1960, considerable mortality occurred in pheasants on some estates in the south of England, due to a respiratory infection associated with the presence of pleuro pneumonia-like organisms. To a lesser extent, partridges, particularly the French partridge, were also affected. The infection was characterized by a nasal and eye discharge and an accumulation of cheese-like material in the infraorbital sinuses. Although this disease has occasionally been observed in previous years, this was the first time it had caused any great concern. It appears very similar, if not identical, to infectious sinusitis in turkeys. The possibility that a virus is also involved has not yet been entirely ruled out.

Canine distemper

There is scant information concerning confirmed cases of canine distemper in free-living wild animals, although Hagan (1943) mentioned its occurrence in wolves, foxes and mink. Robinson, Newberne and Brooks (1955) described an epizootic in raccoons in Indiana, United States, and the causal virus appeared identical to canine distemper virus.

Helmboldt and Jungherr (1955) have recently described the occurrence of the infection in foxes, raccoons and skunks in Connecticut, United States, but it seems probable that in the past many investigators have confused several diseases having distemper-like symptoms with true canine virus distemper. Precise laboratory confirmation is lacking in most cases.

Swine fever (hog cholera)

As might well be expected, the only free-living wild animals which have been shown to harbor this virus are wild pigs, such as in the U.S.S.R. (Saburov and Sokk, 1937), and in the southwest of Germany (Englert, 1953 and Hutter, 1953). These latter workers consider wild pigs as a potent source of swine fever virus in Germany. African warthogs and bush pigs are susceptible to African swine fever, which differs from the disease found in other parts of the world. This disease appears to be mild but domestic pigs contracting the infection can become severely affected. This is another interesting example of the variation of virulence of an infective agent according to the particular host. A description of the early history of the disease in warthogs and their susceptibility to the virus is given by de Tray (1957).

Rinderpest

Carmichael (1938) has furnished most of the available information regarding the feral hosts of rinderpest virus, and it is now apparent that a variety of game animals in Africa can carry the virus, i. e., buffalo, waterbuck, reed buck, bush buck, eland, kudu, duiker and wild pigs, and Guyaux (1951) also stressed the importance of big game animals, especially the buffalo, as reservoirs of this infection for domestic stock.

The annual reports of the colonial territories indicate that much useful work has been done in recent years to elucidate the interrelationships of game and domestic animals in the epidemiology of this disease.

African horse sickness

Horse sickness is well recognized on the African continent and has apparently occurred in free-living zebras (Hagan, 1943). Recently, there has been considerable spread of this disease to other countries, and it is interesting to note that even Cyprus has been affected. The infection tends to "hop" over long distances, even over 1,000 miles, as from Quetta in West Pakistan to Jaipur in India. Consequently, the possibility of reservoir hosts for this virus must be considered.

Malignant catarrhal fever

This disease probably exists in the African buffalo and has been confirmed in the black and blue wildebeest which, however, showed no symptoms, although Mettam (1936) found them to be sources of virus and suggested that "snotziekte" was probably transmitted from wildebeest to cattle.

Yellow fever

The problems associated with the epidemiology of yellow fever both in South America and in Africa are discussed extensively by Dick (1953), who also lists a considerable number of references. Briefly, according to Dick, the position appears to be that an independent sylvan cycle can exist concerning only monkeys and mosquitoes, as first postulated by Balfour as long ago as 1914. Investigation of the forest cycle by Soper (1936 a, 1936 b) showed neutralizing antibody in the blood of monkeys in the areas where yellow fever was present, whereas those from areas where the disease was absent had no antibody. Apparently all species of South American monkeys may possess acquired immunity. Monkeys

are probably the most important source of virus in nature, but there is now some evidence that opossums may also possess antibody. The forest cycle appears to be enzootic, but under certain conditions, epizootics can arise. These epizootics can spread with great rapidity and frequently dead monkeys are found very soon before human cases appear. There is as yet no evidence that birds with viremia can transport the infection from one point to another.

In Africa, there is also a sylvan cycle as well as the man-to-man cycle, and forest antibody surveys have shown that many monkeys are immune. Antibody has also been found in bush babies in Uganda and these may be even more important feral hosts than monkeys in certain areas. During the yellow fever studies, other virus infections, hitherto unknown, have been recognized in free-living monkeys (Dick, 1953). So far little or nothing is known of their epizootiology.

Equine encephalomyelitis

Ten Broeck, Hurst and Traub (1935) suggested the possibility of hosts for the virus of equine encephalomyelitis, quite apart from the horse. This suggestion was based mainly on a study of the general epizootiology of the disease. Subsequently, the naturally occurring disease was confirmed in pheasants (Tyzzer, Sellards and Bennet, 1938) during the 1938 outbreak in Massachusetts and Connecticut, United States. Again, in 1941, Cox, Jellison and Hughes found a naturally infected prairie chicken in North Dakota, where the disease was at that time prevalent in horses and in man. Gwatkin and Moore (1940) examined the brains of ground squirrels and suggested that they might also act as reservoirs. A variety of wild birds has now been shown to be susceptible to experimental inoculation.

The western type of the virus was detected in mosquitoes (*Culex tarsalis*) in nature in 1941 by Hammon *et al.*, and it is interesting to note that this species commonly feeds on wild mallard ducks in addition to other hosts. The accumulated evidence now shows that the disease appears to be spread by mosquitoes which have ingested blood from animals in the early stages of the disease.

Burnet (1953) also drew attention to the possibility of wild birds being an important source of encephalomyelitis virus, and Beaudette

(1951) described the differentiation of virus infections in birds. Marchetti (1949) suggested that virus infection in nutria might bear some relationship to equine encephalomyelitis in South America; several recent records of the recovery of the virus from birds have become available, such as the eastern type of the virus (E.E.E.) from the blood of a purple grackle, an apparently healthy adult, in the United States (Kissling *et al.*, 1951), and the western type of the virus (W.E.E.) from nestling wild birds, such as the magpie and redwinged blackbird, in Weld County, Colorado (Sooter *et al.*, 1951). Antibodies have been detected in wild birds. Sooter, Howitt and Gorrie (1952) point out that during 1949, 11.6 percent of the birds they examined in the Kansas and Missouri areas of the United States showed the presence of W.E.E. antibody. They stressed the fact that whereas the W.E.E. infection causes no apparent clinical disease in wild birds, the E.E.E. type can frequently be fatal.

Wild birds have also been experimentally infected with western equine and St. Louis encephalomyelitis viruses (Hammon, Reeves and Sather, 1951), and W.E.E. virus has now been recorded from naturally infected English sparrows in New Jersey (Holden, 1955). A general review from 1947 to 1951 of equine encephalomyelitis infection in pheasants which, however, had been reared mainly on game farms and could not be considered truly free-living, is given by Beaudette *et al.* (1952).

Further evidence concerning birds as winter hosts for E.E.E. virus was presented by Kissling and his co-workers (1957).

It would seem, therefore, that the principal source of the viruses associated with equine encephalomyelitis and the human disease is a variety of free-living wild hosts, mainly avian, in the various enzootic areas. It may well be, as is so often the case with this group of viruses, that the infection of horses and man is purely a biological accident and is of no importance as regards the survival of the virus.

Infectious porcine encephalomyelitis

Infectious porcine encephalomyelitis, commonly called "Teschen disease," has not so far appeared to be of much significance in wildlife, although Babik (1950) has apparently encountered it in wild boars in Czechoslovakia. He stated that the disease had greatly reduced the herds of wild boar in the affected district.

Louping ill

Although Darling (1947) mentions that the Scottish red deer may be affected with louping ill, there is as yet no clear-cut proof that the clinical disease occurs in this species. It is therefore quite conceivable that the infection in deer might consist of a transient viremia without paralytic symptoms and that the deer might act as an unsuspected source of virus. Dunn (1960) has now reported the occurrence of specific antibody in the blood of deer from various parts of Scotland, and further work on the epidemiological aspects of this problem would be well worth while. The field vole has been shown susceptible to experimental infection and this species might also serve as another natural host for the virus.

Russian spring-summer encephalitis

Soloviev (1941), working in the U.S.S.R., has examined material from many of the smaller wild animals and has confirmed evidence of spring-summer encephalitis (S.S.E.) virus infection in the hedgehog, redgray field vole and mole. Three of four viruses isolated were S.S.E. virus. The carriage of this virus is probably a natural phenomenon in these and other wild mammals, and the infection of man purely accidental from exposure to tick bite in the enzootic areas. Further evidence concerning the detection of the virus of tick-borne encephalitis of man is supplied by Chumakov *et al.* (1940), who isolated strains of virus from hares, squirrels and chipmunk in the Urals.

Kyasamur forest disease

In 1957, subsequent to reports of monkeys dying in the forest, a new tick-borne virus was identified which seemed closely related to the agents which cause Russian spring-summer encephalitis and Omsk hemorrhagic fever. A program of vaccination of people in Mysore State, India, has now been initiated.

Semliki forest virus

This agent was first isolated from mosquitoes in Uganda and is now believed to be also present in the red-tailed monkey.

Mengo virus

The rhesus monkey and mongoose appear to be the natural hosts for this virus in Uganda.

Coxsackie virus infection

Although in a comprehensive survey Tobin (1953) states that man appears to be the only host to the coxsackie viruses, antibody has been detected in normal monkeys (Kraft, 1952), and it is probable that these animals may constitute a widespread reservoir of infection in nature. More recently, O'Connor and Morris (1954) recovered a type of coxsackie virus (Texas I strain) from the blood of a wild cotton-tail rabbit trapped in a nature reserve in the United States. Diluted blood samples from 18 rabbits were inoculated intracerebrally into unweaned mice and one sample produced fatal results. The virus was also recovered from sewage contaminating the area.

Avian encephalomyelitis

Although epidemic tremor is recognized in New England and the adjoining states in the United States, it is not widespread elsewhere; therefore, the preliminary observations of Jennings (1954), that titmice in the United Kingdom may show signs of encephalitis, are worthy of further investigation and transmission experiments. There is little doubt that noticeable mortality does occur among titmice in certain seasons and this may be one of the still undetermined causes.

Epizootic encephalitis in wild ducks in the United States

According to Rosenow (1943), a neurotropic streptococcus and a virus may be associated with epizootic encephalitis in wild ducks, but so far no further confirmatory evidence has become available.

Murray valley encephalitis (Australian X disease)

Although investigations are still proceeding, it now seems clear that this disease is fundamentally an infection of certain wild fauna, especially water birds, in the endemic areas in Australia. Thus Anderson (1953, 1954), working near Mildura and in north-central Victoria, detected neutralizing antibody, by means of the baby mouse intraperitoneal technique, in 40 of 99 free-living water birds. Among the species providing positive specimens were native hen, dusky moorhen, coot, white-faced heron, nankeen night heron, musk duck, black swan, little pied cormorant, little black cormorant, big black cormorant, darter and wood duck. Of the 60 land birds examined, antibody was found in red-backed parrot, white-plumed honey eater, willy wagtail, black-faced wood swallow, white-winged chough, gray shrike thrush and whistling eagle. Miles and Howes (1953), in South Australia, independently detected antibody in ducks, swans, water hens and cormorants. Anderson also detected antibody in the brushtailed opossum and the fox and demonstrated that maternal antibody was transmitted to the immature little pied cormorant. Therefore, there can be little doubt that this infection is primarily a disease of wild birds, especially those frequenting the vicinity of water, and that as the disease has been proved experimentally to be a viremia, it can probably be transmitted from bird to bird and to other incidental hosts such as man and the domestic animals by biting insects. Widespread subclinical infection is likely in man, with occasional severe or fatal cases of encephalitis. This work is, in the present writer's opinion, of great value, in that it pinpoints a virus common in wildlife as the cause of a disease of the central nervous system in man, which is naturally of considerable public health significance.

Infectious encephalitis in foxes

Infectious encephalitis occurs in wild foxes and wolves in North America and apparently bears no relationship to canine distemper. It is caused by a filterable virus and the pathological picture usually consists of hemorrhages in the central nervous system and viscera. Perivascular infiltration with round cells in the central nervous system is commonly present. Epizootic fox encephalitis has been recorded by Green *et al.* (1930). Bezdek (1942) described encephalitis in the red fox

in southwest Ohio, and Green, Evans and Yanamura (1943) mentioned the susceptibility of raccoons to this virus.

Bilirubinemia and jaundice in raccoons

An agent causing convulsions, bilirubinemia, jaundice and death in raccoons, has been isolated by Kilham and Herman (1954) in Maryland, United States. They consider this infective agent to be a virus because it is filterable, and examinations for bacteria and *Leptospirae* have proved negative. It appears to be closely allied to the virus of canine distemper.

The main characteristics of the disease appear to be conjunctivitis and an elevated serum bilirubin, with or without jaundice.

Dengue

This virus, which shows the characters of the arbor viruses, causes widespread disease in the tropics. Monkeys are probably involved in the infection cycle.

The transequitorial relationships of the dengue fevers was discussed recently by Rowan and O'Connor (1957). The demonstration of antibody in migrant wading birds in North Queensland supports the theory of this relationship.

Japanese encephalitis

Tabuchi, Hosoda and Narita (1951) examined brain material from migratory wild birds for the presence of virus, without success, but eventually isolated an infective agent from ectoparasites on these birds, which was classified as Japanese encephalitis virus; precise information on reservoir hosts is, however, still lacking. It is interesting to compare the views of these workers with Anderson's findings in Murray valley encephalitis (1953, 1954), which appears to be a very similar disease.

" West Nile " encephalitis

Evidence suggests that the causal virus which is similar to Japanese B virus is present in wild birds in the affected areas.

B virus

B virus infection appears to be endemic in monkeys, causing a mild, rarely fatal disease. Infection in other species, including man, is frequently fatal. Cases in man have arisen from the bites of rhesus monkeys.

Lymphocytic choriomeningitis

This disease is common in house mice, often reaching endemic proportions, and cases in man usually arise from this source.

4. DISEASES DUE TO PROTOZOA AND CLOSELY ALLIED ORGANISMS

Leishmaniasis

According to Hoare (1955), leishmaniasis is one of those interesting conditions in which the lower mammals play an important role as reservoir hosts. The disease in the human subject can be one of two types, either cutaneous, i. e., oriental sore, or Kala-azar, affecting the viscera. The infection is widespread from India and China to the U.S.S.R. and also in those countries bordering on the Mediterranean. Much of the research concerning the epidemiology of the cutaneous variety has been done in Soviet Middle Asia, and two types have been described, a chronic urban form, and an acute moist rural form, with rapid ulceration (Kojevnikov, 1941, 1942; Latyshev and Kriukova, 1942).

The incidence in human beings is most marked in residents or visitors to remote rural districts, and sandflies (*Phlebotomus*) from the burrows of certain rodents, such as gerbilles, carry the infection. The rodents were found to have typical sores on their ears and noses and *Leishmania tropica* was recovered from them. Hoare (1955) describes a striking control experiment in a desert area in Turkmenistan, where 70 percent of the local population had moist desert sores. Chloropicrin poisoning of the burrows in the area was most effective because of the limited area of distribution of sandflies from their breeding haunts, and a year later the infection had been practically eliminated from the human population in the area. The moist type of disease also occurs in Iran, and again gerbilles are the main reservoir hosts (Ansari and Faghih, 1953). It is likely that a similar situation exists in northern and western Africa. In the New World, del Ponte (1952) suggested that the agouti is the main reservoir for " Espundia " (*L. braziliensis*) in the forests of Paraguay.

Latyshev *et al.* (1951) found that Kala-azar in Middle Asia was associated with spontaneous visceral leishmaniasis in jackals, thus providing natural infected hosts for sandflies. Foxes are also likely hosts in this area, and in Brazil foxes are known to be naturally infected with *L. donovani*. Heisch (1954) has provided some evidence to show that wildlife hosts are also present in Kenya.

Trypanosomiasis (sleeping sickness and Chagas' disease)

In man, trypanosomiasis is known as sleeping sickness in tropical Africa and Chagas' disease in Central and South America.

Sleeping sickness may be one of two types, i. e., chronic (Gambian) or acute (Rhodesian). The causal agent may, in fact, be the same trypanosome, one strain being more virulent than the other, and both are indistinguishable morphologically from *T. brucei*, which infects farm stock. The question of reservoirs is not yet fully understood (Hoare, 1948); interinfections do, in fact occur, and tsetse flies, domestic or wild mammals may be infected with any of these trypanosomes. There is considerable difficulty, therefore, in determining the precise species and often this eventually depends on epidemiological data. It is considered by some workers that the Rhodesian disease in bush areas is transmitted to man from antelopes, and vice versa, and that antelopes may be very important reservoir hosts for this class of trypanosome, although there has been some divergence of opinion on this particular point (Corson, 1936; Duke, 1936; Fairbairn, 1948).

According to Hoare (1955), in the New World the role of the lower mammals in Chagas' disease is clearer. Many wild animals are infected with *T. cruzi*, and the vectors are triatomid bugs associated with armadillos and opossums, the chief reservoir hosts. The first case of the disease in man in the United States was recorded by Woody and Woody (1955); wood rats and their associated bugs are naturally affected with *T. cruzi* in the Southern States.

In South and Central America, trypanosomes, indistinguishable from *T. rangeli*, have been recovered from Cebus monkeys and opossums, which are probably the main reservoir hosts (Hoare, 1953; Pifano, 1954; Floch and Fauran, 1954; Zeledan, 1954).

Hoare stresses the fact that often the wild reservoirs are symptomless carriers because of the stable host-parasite relationship in primitive en-

zootic foci. Thus these foci in nature may not at first be detected, and consequently they are always a potential epidemiological danger to human beings entering the area.

Trypanosomiasis in game animals

Nagana

Trypanosoma brucei causes nagana or "fly disease" in nearly all parts of tropical Africa, such as the Sudan, Uganda and Tanganyika. It occurs in Rhodesia, but there it is rare compared with *T. congolense* and *T. vivax* infections. The parasite is transmitted by the tsetse fly (*Glossina*), and it has been shown that wild animals, particularly the younger age groups in the affected areas, can harbor this and related parasites in small numbers, sometimes without any pronounced pathogenic effect. Because of this, trypanosomes can survive in these "fly belts," independent of domestic stock. It is interesting to note that game of the same species from tsetse-free areas may succumb rapidly to the experimental inoculation of trypanosomes. A small trypanosome (*T. congolense*), which infects cattle, horses, pigs and dogs in tropical Africa, is also present in most species of wild game animals, and *T. vivax* is commonly found in antelopes in addition to several other hosts. Antelope probably constitute the principal natural hosts for this particular parasite, which can cause infection of most species of domestic animals in the tsetse fly areas. Much of the work on pathogenic trypanosomes in the larger species of wildlife has been done in Eastern Africa, where the role of game as reservoirs of infection is well recognized. The recent isolation of *T. rhodesiense* from bushbuck and hartebeest is of particular importance.

General information regarding the importance of big game in the general epidemiology of trypanosomiasis is available from papers by Bruce *et al.* (1914), Buchanan (1929) and Vanderplank (1942, 1947), and also in *Protozoology* by Wenyon (1926); these workers describe the identification of different trypanosomes from a variety of game, such as elephants, giraffes, warthogs, kudu, hartebeest, impala, eland, duiker, bushbuck, reedbuck and waterbuck. Buxton (1955) has produced a most comprehensive work on the natural history of tsetse flies. He points out that "fly disease" is probably more important than any other Afri-

can disease and that this may, in part, be responsible for the lack of foreign intrusion in the past.

From the limited amount of information available, the question arises as to whether much of the game destruction policy advocated in Africa for the control of trypanosomiasis has been based on sound scientific fact. Research has been lacking with regard to the determination of possible alternative hosts for the tsetse fly, quite apart from the well-recognized ones among the larger game animals. The only scientific experiment on game destruction (Potts and Jackson, 1952) showed that the method is successful in eliminating certain species of tsetse, but also pointed out the undesirable features of game destruction. Some interesting work has been done on the serological identification of blood meals from *Glossina morsitans* (Weitz and Jackson, 1955). From the methods used by these authors, the hosts on which these insects have fed might readily be identified and a conclusive answer obtained on the real significance and importance of the different species of big game in trypanosomiasis.

Surra

Surra, caused by *Trypanosoma evansi*, occurs in India, Burma, Ceylon, south China, Thailand, Sumatra, Java, the Philippines, Madagascar, Iran and Arabia. Camels in the Sudan, Algeria and the United Arab Republic also suffer from surra, but the disease appears to be mild in character.

Reservoir hosts in wildlife are principally the water buffalo and other ruminants in Asia; it is recognized that camels can apparently become chronic carriers in some areas.

Losses frequently occur in horses which have been brought into enzootic areas near herds of water buffaloes. Transmission is effected mainly by *Tabanidae* or "horse flies," but the vampire bat has also been known to transmit this disease in the Panama region of America. It is considered possible that the bat serves as a carrier between cattle and wildlife hosts, which show no symptoms, and horses, in which the disease is serious.

Mal de Caderas

Mal de Caderas is known to occur in Brazil, Bolivia, Paraguay and Argentina. The causal agent is *T. equinum*. Horses are principally

affected and transmission takes place by flies of the *Tabanidae* and *Stomoxys* groups. It is considered that the capibara, a rodent, resident in the vicinity of the rivers, is probably the main natural reservoir of infection. The disease may be fatal in this species, which often suffers from severe epizootics, during which a trypanosome, indistinguishable from *T. equinum*, has been demonstrated in them.

Murrina

Murrina, often known as "derrengadera," is a disease of equines attributed to *Trypanosoma hippicum* in the Panama Canal zone, and possibly Colombia. The native burro appears to be an important reservoir, and this species, as well as deer and wild hog (peccary), can be experimentally infected.

Trypanosoma venezuelense

Trypanosoma venezuelense causes a disease of horses and dogs in Venezuela, in many ways similar to surra. This trypanosome closely resembles *T. evansi* and *T. hippicum*, the parasites causing surra and murrina, respectively, and may not be a separate species. As long ago as 1905, Rangel considered that the wild dog, capibara, and a species of monkey represented the natural reservoirs of infection.

Trypanosomiasis of rats

Trypanosoma lewisi has been found in free-living rats in practically every country throughout the world. The rats generally show little inconvenience from the infection, although some strains of the parasite do, in fact, appear to be somewhat more pathogenic than others. Various types of rat fleas are considered to be the main transmitting agents. A similar trypanosome is associated with gerbilles in South Africa.

Trypanosomiasis of birds

Although, according to the literature, trypanosomiasis is common in over 200 species of wild birds, frequently being detected in nestlings only a few days old, it is very rare in any of the domestic species.

Many of the trypanosomes found in birds have, in the past, been named according to their individual hosts, and it is possible that arthropods are commonly involved as vectors in many cases. The degree of pathogenicity of the trypanosomes varies, but the majority appear to be quite harmless to their hosts and disease is rarely encountered in them. It is interesting to note that frequently the bone marrow of wild birds can be shown to be infected, although the blood is negative and there also appears to be a seasonal fluctuation in the number of trypanosomes present in the blood.

Other evidence of trypanosomiasis in wildlife

The cotton rat has been shown to carry trypanosomes in Florida, United States, and field mice have now been mentioned as suitable permanent carriers of trypanosomes (Werner, 1949), although other workers have previously suggested this possibility. *Trypanosoma gambiense* has been isolated from bush babies in captivity after ingestion of infected rats (Heisch, 1952). All these observations point to the ever-increasing significance of various small wild mammals acting as reservoir hosts for certain trypanosomes, quite apart from the big game species. Several different trypanosomes have been described in bats examined in many countries, but the true significance of these findings is not yet fully determined. The mole and shrew have also been shown to carry trypanosomes, and in all these cases it is probable that arthropod vectors are involved.

Trichomoniasis

A disease of young pigeons caused by *Trichomonas gallinae*, also known as *T. columbae*, was first described by Rivolta in Italy in the nineteenth century. More recently, in the United States, Cauthen (1936) found the disease in ringdoves and mourning doves, but these could not be considered as being truly in the wild state. Subsequently, Stabler (1937) examined over 100 pigeons and many other birds and found trichomonads in 82.4 percent of the pigeons and in 8 hawks. Haugen (1952) found trichomonads in a widespread outbreak in Alabama mourning doves during 1950. The disease was considered responsible for recent

decreases in the dove population. Callender and Simmons (1937) also reported the infection in Java sparrows, Tovi parakeets and Vorraux's doves in the United States, and there is every likelihood that the disease may be widespread in those species in nature. The disease usually involves the vicinity of the mouth, throat and esophagus, resulting in cheesy-like lesions. Frequently, infected birds are extremely emaciated.

It is generally considered that pigeons are the principal hosts for this parasite, and it is the author's experience that many wild wood pigeons in the United Kingdom are affected. In the past, it has probably been frequently confused with pigeon pox in this species. During the wood pigeon survey work at Oxford in recent years, many squabs collected from the nests of free-living wood pigeons died shortly after admission to the laboratory as a result of trichomoniasis. The evidence clearly pointed to a previously acquired natural infection, probably during the act of feeding in the nest. Young pigeons dying from this disease frequently show necrotic lesions in the lungs and liver. It is considered to be a serious disease in domestic pigeons.

Other flagellates, such as *Chilomastix*, are commonly found in the alimentary tract of a variety of species of wild duck, as well as in rodents, such as gerbilles, rats and mice, but they appear to be of little or no significance insofar as the production of disease is concerned. *Hexamita meliagridis* has, however, been shown to be associated with enteritis in quail and chukkar partridges by McNeil, Platt and Hinshaw (1939). In Europe, sterility in roe deer in the forests near Kassel has been said to be related to the presence of trichomonads in the genital tract (Schoop and Stolz, 1939).

Histomoniasis (blackhead)

Disease due to *Histomonas meliagridis* has been recorded in pheasants (Wenrich, 1941); black grouse (Salhoff, 1938); quail (Hagen, 1943). It is also well known in partridges in the United Kingdom, and these natural hosts can constitute important sources of infection for domestic turkeys in enzootic areas.

It is interesting to recall that turkeys were originally native American birds, and it is quite conceivable that *Histomonas* has been distributed to many countries throughout the world from original foci in Mexico. It is probably true to say that the disease is now present wherever

turkeys are reared. It is well recognized that a common nematode, *Heterakis*, is an important factor in the transmission of the disease. This nematode is present in most of the wild species of birds known to be susceptible to blackhead.

In recent years, blackhead has become one of the major problems in the management of wild partridge stocks in the United Kingdom, and many cases have been encountered in this species by the author and also by Dr. P. Clapham in the south of England. Wilson (1952) has also confirmed the existence of the disease in partridges in Scotland. The disease affects the partridge in much the same way as the turkey, causing marked necrotic lesions in the liver and caeca. On certain estates in the south of England, blackhead has been blamed for most of the mortality in partridges in recent seasons, and there is little doubt that in some areas the native partridge population must constitute a severe potential hazard to the introduction of domestic turkeys into those frequently remote districts, areas which would otherwise appear to be most suitable, because of the scarcity of domestic fowl.

So far in the United Kingdom, apart from the native gray partridge and the pheasant, no infection has been detected by the author in any other species of gamebird, including the redleg or French partridge.

Coccidiosis

Coccidia have been detected in practically every species of vertebrate and invertebrate, and as these protozoa apparently possess a high order of host specificity, infections are rarely contracted from other species. Moreover, no cross-immunity exists between species. With rare exceptions, the presence of most of the species of coccidia appear to cause little or no inconvenience to their hosts and may almost be considered as part of the normal intestinal population.

Isospora rivolta

Isospora rivolta is relatively harmless and rarely causes disease, although several wild animals have now been shown to carry it. A variety of species of coccidia from free-living wild mammals has been described (Salhoff, 1939) in roe deer in Germany and in bighorn sheep in the United States (Honess, 1942). Yakimoff and Matschoulsky (1935)

have recorded the isolation of coccidia from bears, wolves and wild dogs, and Yakimoff *et al.* (1939) have encountered coccidiosis in reindeer. The exact significance of many of these records from the pathological viewpoint is, however, difficult to assess.

Coccidiosis of rabbits

Several types of coccidia occur in rabbits, and of these two are common and definitely pathogenic.

Eimeria stiedae

Eimeria stiedae was probably the first coccidium known. It is extremely common in young wild rabbits, producing the well-known characteristic white or yellowish lesions in a greatly enlarged liver. It is probably true to say that in certain seasons in the United Kingdom, practically every young rabbit examined shows evidence of a past or present infection. The exact mortality in the infected wild rabbit population is not known, but infected rabbits appear to be capable of surviving, despite considerable liver damage. Sometimes severe enteritis is seen, caused by another species of coccidium. Clapham (1954) drew attention to the widespread, severe infection of rabbits and hares in the United Kingdom during the winter of 1953 and pointed out that it was possible that much of the mortality associated with coccidiosis was being wrongly attributed to myxomatosis. Intestinal coccidiosis is generally produced by *Eimeria magna* and possibly *E. viresidua.* Mortality in mountain hares in Scotland was also attributed to coccidiosis by Ritchie (1926). Further information on the coccidia of wild cottontail rabbits in the United States was supplied by Honess (1939), and Carvalho (1943) gave a useful description of coccidia in the wild rabbits of Iowa and attempted cross-infection of the domestic species. It has also been said that coccidiosis is one of the diseases responsible for mortality in gray squirrels in the United Kingdom, but precise scientific proof on this point is still lacking; it is the author's opinion that many deaths in gray squirrels are, in fact, due to *Capillaria*, a nematode which shows a predilection for the liver tissue. Apart from domestic rabbits, it is generally believed that domestic animals are not susceptible to coccidia found in any wild mammals.

Coccidiosis in birds

At least eight species of coccidia occur in domestic fowl, and at one time it was believed that coccidiosis in domestic poultry could be spread from one flock to another by wild birds. This was disproved, however, by Smith and Smillie (1917), who found that although sparrows commonly carried coccidia, they were of the *Isospora* genus. The commonest species in sparrows and other wild birds is *Isospora lacazii*, which is apparently of little or no significance as a disease agent in domestic birds, except perhaps in certain small aviary birds, such as canaries and some types of finches. Experimental chronic *Isospora* infection of sparrows in the United States was discussed in detail by Boughton (1937).

Brinkmann (1926) was probably one of the first observers who found that disease in wild game birds was caused by coccidia, namely, in the Norwegian willow grouse. *Eimeria phasiani*, *E. dispersa* and *E. tennela* may all occur in pheasants, but are apparently not very pathogenic (Richardson, 1948). The existence of coccidiosis has been confirmed in sage grouse (Honess, 1942) and in quail (Herman, Jankiewicz and Saarni, 1942) in the United States. Coccidiosis is recognized as a common disease of partridges in the United Kingdom, causing a marked enlargement of the caeca, but it rarely appears on well managed estates. It seems to arise chiefly as a result of overstocking in a limited area. In the U.S.S.R., Yakimoff and his co-workers (1935, 1939) have described in numerous publications the isolation of coccidia from a variety of wild birds, but there does not seem to be much evidence pertaining to the actual production of disease.

A useful review of the coccidia from the avian orders Galliformes, Anseriformes and Charadriformes is now available (Levine, 1953).

A really comprehensive survey has long been required of the coccidia in free-living wild animals, with a view to determining the precise significance of these parasites and their possible harmful effects on the different varieties of domestic stock.

Hemoproteus, Leucocytozoon, Plasmodium

Members of these genera occur, without apparent ill effects, in innumerable wild birds, hundreds of species having been listed as hosts, and it is believed that the transmitting agents are mainly blood-sucking flies.

Leucocytozoon

Leucocytozoa are found only in birds and are of common occurrence in many species. On rare occasions they are said to cause heavy losses in domestic ducks and turkeys and also in a variety of wild duck, wild turkey and grouse. In some cases, the transmitting agents have been shown to be flies of the family *Simuliidae.*

It is claimed that *Leucocytozoon simondi* can attack both wild and domestic ducks and that mortality may be severe in young birds. Adult ducks can sometimes act as carriers (O'Roke, 1930, 1931, 1934). Affected birds may show signs of stupor; there appears sometimes to be some difficulty in maintaining equilibrium and purulent conjunctivitis may be present. Hemorrhagic enteritis may be found on post-mortem examination.

Clark (1934) demonstrated *L. bonasae* in ruffed grouse dying in large numbers in Ontario. Again, mainly the young birds were affected, and by midsummer upward of 60 percent had died. All the dead birds examined were shown to have the parasite in their blood. The same author suggested that this disease may be mainly responsible for the periodic heavy mortality in that area, resulting in marked fluctuations in the grouse population. Further contributions on this subject have been made in later publications by the same author (Clark, 1935 a and b). It is interesting to note, in view of the above findings, that Borg (1953), on the contrary, working in Sweden, did not find *Leucocytozoa* responsible for deaths or disease in Swedish forest game birds, such as the capercaillie, black grouse and hazel grouse, although he found a high rate of carriage in all three species. In his report, he gave a very comprehensive list of references pertaining to this subject in various countries. The finding of *Hemoproteus* in the blood corpuscles of Moroccan birds is recorded by Gaud and Petitot (1945), and Herman (1935, 1944) gives further general information on the blood protozoa of American birds. The exact significance of all these findings is, for the present, difficult to assess, but the general concensus of opinion appears to be that these protozoa may be considered as potential pathogens and, under certain exceptional conditions, may be the cause of considerable mortality in free-living wild birds.

Malaria

Malaria, caused by species of the genus *Plasmodium* is, without doubt, one of the most widespread and serious diseases of man. Among

wild, free-living animals, plasmodial infection is known to occur in monkeys, squirrels, rats, shrews, bats and the flying fox in the Congo and in Australia; nevertheless, birds are the most frequent and important hosts. Generally, the parasites are host specific, but this is not always the case in the bird malarias. At least 30 species of North American birds probably harbor malarial parasites but, although many bird-infecting strains of *Plasmodium* have been described, it is probably true to say that this far exceeds the actual number of species involved. Manwell (1938) limits them to about 12, 9 of which he considers valid species; Herman *et al.* (1954) stated that in a survey of 8,674 birds of 73 species in the United States, 7 species of *Plasmodium* were found in 888 specimens of 27 species. *Plasmodium relictum* was demonstrated in 79 percent of the infected birds. The inoculation of blood from the wild birds into canaries demonstrated many latent infections which were not readily demonstrable by direct smears.

In the past, much information has accumulated from the study of avian malarial parasites which has proved of great value in the general study of human malaria. The avian malarial infections are also transmitted by mosquitoes but, unlike the human disease, they are generally carried by *Culicine* species.

Plasmodium praecox, *P. circumflexum* and *P. cathemerium* are relatively common in passerine birds in North America, although in nature they do not appear to be highly pathogenic. They have been studied in recent years in some considerable detail, both in free-living and in captive specimens.

Wetmore (1939) recorded species of *Plasmodium* in the sharptailed grouse and discussed the possibility of such infections being transmitted to other species of birds. Other interesting contributions include *Plasmodium* infection in penguins (Rodhain and Andrianne, 1953); in doves and pigeons (Coatney, 1937); in red-winged blackbirds (Herman, 1937); and a general article on malarial parasites in the birds of Greece (Papadakis, 1935).

Several general reports have also appeared during the last few years concerning a variety of blood parasites in birds in different countries, especially in the United States (Huff, 1939; Nelson and Gashwiler, 1941; Wetmore, 1941; Jordan, 1943 and Thompson, 1943). Further information has been contributed by Hewitt (1940) and by Beltran (1940) concerning Mexican birds. Similar information is available from South

Africa (Enigk, 1941). Morello (1938) has described several types of blood parasites in birds of prey.

Very little has been done in the United Kingdom to investigate the problem of malarial parasites in wild birds, and the original paper by Coles (1914) can be cited as the main British contribution to the literature on this subject. This field, so far as the United Kingdom and many other countries are concerned, is still virtually untouched.

Theileriosis

According to Richardson (1948), apart from domestic stock, theilerial infections have been detected in various mammals such as eland, hartebeest, duiker, bush buck, kudu, waterbuck and warthog. The exact relationship between these game parasites and cattle has not yet been determined. The majority of the *Theileria* have been found in apparently healthy animals which have been shot. The African buffalo and Indian water buffalo also appear to be susceptible to true East Coast fever infection, although the disease in them may be of a mild nature.

Neitz, Canham and Kluge (1955) drew attention to the discovery of a fatal form of tick-transmitted theileriosis in cattle in Zululand, which was apparently contracted in the Corridor, a stretch of country between two game reserves. There appears to be little doubt that buffaloes or perhaps some other variety of game are the reservoir hosts for this infection. As the disease is quite distinct from that produced by *Theileria parva*, it has been provisionally recognized as "corridor disease," and the causal agent has been named *Theileria lawrencei*. The occurrence of theileriosis in Australian wild animals is recorded by Seddon (1952).

Piroplasmosis and anaplasmosis

The southern blue tick (*Boophilus annulatus*) appears to be the main transmitting agent of Texas fever in the United States, and it is noticeable that where the eradication of the tick has been accomplished, the disease has disappeared from domestic stock. Cases of Texas fever are, however, known to occur in deer in the United States, and it is highly

probable that reservoir hosts, quite independent of cattle, are present in those other countries where the disease, due to this type of parasite, occurs.

Other cases of piroplasmosis have been encountered in a variety of wild animals, such as in jackals in India, where the same species of *Piroplasma* affects dogs. *Babesia* have recently been detected in the Cape dassie by Jansen (1952) ; *Nuttalia cynicti*, claimed to be a new species in the yellow mongoose in South Africa, was first demonstrated by Neitz (1938), and Heisch (1952) recovered *Piroplasma* from the genet cat and ground squirrel in Kenya.

Anaplasmosis in wild deer, associated with sporadic outbreaks in neighboring cattle, has also been recorded by Boynton and Woods (1940) in California, and Osebold, Christensen, Longhurst and Rosen (1959) have shown the importance of deer as " silent " reservoir hosts, since a well-advanced adaptation of host and parasite exists. It is suggested that the wild ruminant may well be the natural host for the organisms, with cattle playing a secondary role. Several species of bats, in Italy and in several other countries, have been shown to carry piroplasms, and a variety of small rodents such as field mice and water voles in the United Kingdom, the dormouse in Italy, the striped zebra mouse in the Sudan, and the edible rat in Nyasaland, are proven carriers.

Balantidiosis

Balantidium coli infection has been recorded in monkeys on rare occasions, but is apparently of little or no significance as a disease-producing agent under natural conditions.

Toxoplasmosis

In 1909, Nicolle and Manceaux found a parasite in a small mammal, the gondi, an inhabitant of the Sahara desert in northern Africa. They named it *Toxoplasma gondi*, and since the first isolation, parasites indistinguishable from *T. gondi* have been found in a number of species of wild birds, mice, rats and hares. There is some evidence that it also occurs in jackals in South Africa. In one of the earliest records

of this parasite causing disease in free-living wildlife (Findlay and Middleton, 1934), reference was made to a nervous disease and mortality in voles in the United Kingdom, with demonstrable toxoplasms in the central nervous system. It seems probable that this disease may well play some part in the periodic four-yearly cycle in the vole population.

It has been shown that these parasites are not host specific, transmission from one species of animal to another having been accomplished in the laboratory. There is no doubt that many species of toxoplasms which have in the past been classified separately are in fact identical.

Toxoplasma gondi may be found singly or in compact masses in cyst-like structures in practically all the tissues of the body ; an encephalitis is a common finding. The route of transmission is, however, still not completely understood. The disease appears to be common in hares and occurs also in capercaillie and black grouse in Sweden, according to Borg (1953a), who recorded that about 140 cases of toxoplasmosis were found in those two species during 1948-52.

The clinical signs for the disease in capercaillie are often considered almost specific, especially the twisting of the neck on its long axis. Borg in a further communication (1953b) recorded that 48 of 531 mountain hares were found to harbor the parasite, and it was also demonstrated in 60 common hares. It was considered that toxoplasmosis was a common cause of death in all these species. Borg also mentioned the infection in pheasants and in an osprey. Toxoplasmosis in game in Sweden appears to match, geographically, the occurrence of the human disease. In some parts of Sweden the incidence in human beings, based on serological tests, is as high as 45 percent. There is some evidence that insects might be responsible for spreading the disease, and if this is true, the risk to human beings in areas where chronic cases exist among the game birds is not inconsiderable. The first cases in wildlife in Sweden occurred about the same time as the first human infections were detected (Hülphers *et al.*, 1947).

Toxoplasmosis was also described, macroscopically and microscopically, in 12 hares by Studić (1953) in Croatia. In Denmark, Christiansen (1948) recorded the results of post-mortem examinations on 2,411 hares during the years 1935-47 inclusive. All hares had died spontaneously and many presented a pathological picture which Christiansen considered pathognomonic for toxoplasmosis, viz., enlarged spleen (Figure 16), necrotic foci in the liver with subcapsular hemorrhages, enlarged mesenteric lymph nodes, edema and congestion of the lungs and reddish

pleural exudate. He pointed out that the over-all incidence was probably about 9 percent, and that on the island of Bornholm it rose to about 29 percent.

It has been suggested by Weinman and Chandler (1954) that toxoplasmosis in rodents could be a potential human hazard, and this is supported by the fact that the incidence of this parasite in samples of rats in the United States may reach 8 percent. Manwell and Drobeck (1951) discussed the occurrence of mammalian toxoplasmosis in birds in eastern North America, and Herman (1937) also contributed an interesting article on the incidence of toxoplasmosis in North American birds. At least 12 families have been found to be involved ; more recently, Jacobs *et al.* (1952) recorded toxoplasmosis in wild pigeons. In the United Kingdom, Beverley *et al.* (1954) showed that rabbit trappers possessed a high toxoplasma antibody content compared with other members of the population, and although no isolates were made, they also detected antibody in wild rabbits from a small area in Wales. These authors suggested that toxoplasmosis may be more common that has hitherto been believed.

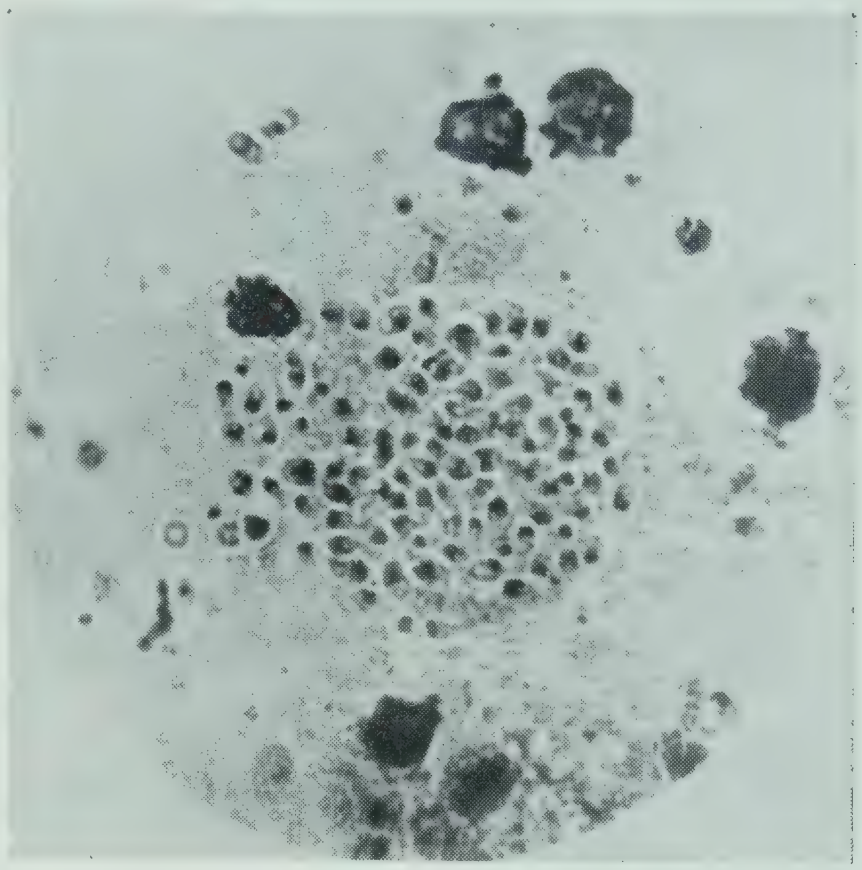


FIGURE 16. — Toxoplasmosis; pseudocyst in the spleen of a hare (Harris-Hematoxylin).

Toxoplasmosis in wildlife is now being recognized in several other countries. Recently, in New Zealand, Australia, and in the United Kingdom, toxoplasma-like bodies have been associated with a type of abortion in sheep, and although it is too early to say if this infection is in any way related to disease in neighboring wildlife species, evidence from Australia tends to support this theory. Because of its obvious importance to man and domestic animals, further investigations into possible reservoir hosts in nature are urgently required.

Sarcosporidiosis

The intracellular parasites of the *Sarcocystis* genus have been encountered in wild herbivorous animals (Hagan, 1943), in rodents such as rats (Farris and Griffith, 1949), and in a variety of species of birds, especially surface-feeding and sometimes diving ducks (Wickware, 1944). They have also been described in swallows (Quortrup and Sudheimer, 1944), and it is interesting to note that in these birds the sarcocysts were present in the muscles of the head and neck, whereas in ducks, chiefly the pectoral region is affected.

These parasites are not always host specific ; Darling, in 1910, transmitted an infection from an opossum to guinea pigs. It is quite conceivable that all the so-called varieties may, in fact, represent a single species. The parasites can be observed in striated muscle of the host and, generally, the animal is not seriously affected. Frequently, no untoward effects are noticed. The actual transmission of the disease is not yet fully understood.

Shillenger and Wetmore (1938) and Quortrup and Shillinger (1941) have described the importance of sarcosporidiosis as a disease of wildlife, particularly wild fowl in the United States, and probably the most comprehensive survey in recent years has been furnished by Scott (1943a and b), who mentions cases in several wild animals as well as in domestic species, and considers the economic importance of the sarcosporidia as well as their life histories.

5. DISEASES DUE TO RICKETTSIAE

Tropical typhus of Malaya and the East Indies

One variety of tropical typhus, known as "shop typhus," is spread by rat fleas, probably *Xenopsylla cheopis*, and rats constitute the main reservoir of infection, with sporadic outbreaks arising from them to human beings. Another form of this disease, known as "scrub typhus," is spread chiefly by mites and is known locally in Sumatra as "mite fever." The present evidence is that rats probably constitute the chief reservoir of infection.

Mexican typhus

The endemic type of typhus, which occurs chiefly in the summer and autumn, especially near seaports, and which has a comparatively low mortality, is also apparently carried by *Xenopsylla cheopis*, with the rat again playing the part of principal host. The elucidation of the role of rats and their fleas was largely accomplished by a group of Mexican and American workers, among whom were Maxcy (1926, 1928, 1929); Mooser (1928); Mooser *et al.* (1931); Dyer *et al.* (1931 a, b, c; 1932 a, b, c); Ceder *et al.* (1931); and Rumreich (1933).

Endemic rat flea-borne typhus has also been found in many other areas of the Old World, i. e., France, U. S. S. R., Manchuria and elsewhere. Examination of wild rats caught in those areas has shown conclusively that they do, in fact, carry murine typhus, and consequently give a positive Weil-Felix reaction to *Proteus* OX 19. Penfold and Corkill (1928); Marchandier *et al.* (1931); Kodama and Takashashi (1931); Kodama *et al.* (1932); Brumpt (1932); Lepine (1932); Lawton and Murray (1933); Kritschewski and Solowiow (1934); Suzuki (1934); and Nicolle and Sparrow (1934) are a few of the numerous references to this particular subject.

Japanese flood fever (tsutsugamushi)

Japanese flood fever occurs in low-lying areas in Japan and the East Indies, mainly during the summer and autumn months. It is caused by the bite of a mite frequently found on field mice and rats, which undoubtedly constitute the principal reservoir of infection, without evidence of clinical symptoms.

The causal agent is *Rickettsia nipponica* (Sellards, 1923), and the case mortality in the human being may be as high as 40 percent.

In many respects the disease is very similar to the scrub typhus of Malaya, apart from the high mortality. The general evidence shows that, if not actually identical, these viruses are closely related (Lewthwaite and Savoor, 1936).

Rocky Mountain spotted fever

Rocky Mountain spotted fever, caused by *Rickettsia rickettsi*, is essentially a disease of North America ; it is tick-borne by *Dermacentor andersoni* or *D. variabilis*, and rats and mice can be infected experimentally, without clinical disease developing (Fukuda, 1929). The disease is different from the louse-borne and murine groups, and from scrub typhus and Japanese river fever, and there is little doubt that wild rodents probably play an important part in the epidemiology of the infection, with man accidentally becoming infected under certain predisposing circumstances.

São Paulo typhus

São Paulo typhus seems in many ways to be very similar to Rocky Mountain spotted fever and also follows tick bites, although in this case the tick appears to be *Amblyomma cajennense*. There is every possibility that wildlife reservoirs exist for this disease.

Trench fever

There is some evidence that quite apart from the cases of trench fever which occurred during the first world war, infection remained as

an endemic in certain countries, such as Poland and U. S. S. R. (Laurell, 1933 and Braslawsky 1934). The possibility that, quite apart from the lice, some wild rodent species might be the principal hosts for the causal agent, *Rickettsia quintana*, cannot be completely excluded, and this problem awaits further investigation.

Q fever

In Australia, one of the principal natural reservoirs of *Coxiella burnetii*, the cause of Q fever, which is important from both the veterinary and public health angles, is the bandicoot, a small marsupial animal, which was shown to possess the specific antibody by Derrick *et al.* (1939). Derrick and Smith (1940) described the isolation of three strains of the rickettsia from the bandicoot, and Pope, Scott and Dwyer (1960) have drawn attention to the role of the kangaroo as a reservoir host in West Queensland. It is probable that there are numerous wildlife reservoir hosts, as yet undetected for this rickettsia, and that the disease is widespread. There is evidence that it is present in ticks as well as in animals in Algeria, Morocco and Portugal, and it is possible that *Coxiella burnetii* is also a natural infection of birds (Babudieri and Moscovici, 1952). It can also be understood how species such as sparrows, starlings and feral homer pigeons could readily acquire infection from the excreta of domestic animals.

So far, however, little has been done to investigate this problem, but with the widespread occurrence of Q fever in the human and domestic animal populations throughout the world, every effort should be made to initiate surveys of wild animal populations, especially rodents and birds. It has been suggested that, by means of the complement fixation test, it should be possible to rapidly survey a variety of species for Q fever, but the value of this test may perhaps be limited, because birds apparently become rapidly negative to an antibody test, although continuing to harbor the rickettsia in their tissues.

Rickettsial pox

In the human subject, rickettsial pox is characterized by a papulovesicular rash and the development of a remittent febrile reaction, three

to ten days later. The causal agent, *Rickettsia akari*, has been said to be closely related to the rickettsia of the spotted fever group. Fundamentally, it is essentially a disease of house mice, which are also infected with mites (*Allodermanyssus sanguineus*) which apparently act as vectors, and human beings in large cities such as New York (Huebner, 1950) acquire infection accidentally from this source.

Heartwater

Heartwater is of some considerable importance in cattle, sheep and goats in south and central Africa. There is little doubt that reservoirs of *Rickettsia ruminantium* exist in several species of wild game, and Neitz (1935, 1944) has confirmed the existence of the disease in springbuck under natural conditions and has also demonstrated the susceptibility of blesbuck and black wildebeest. A giraffe has also been known to die from heartwater in the game park at Livingstone. Jackals and Cape hunting dogs are said to be reservoirs of *R. canis* in South Africa.

Tick-borne fever

Although no wild host has yet been recorded for the causal rickettsia of tick-borne fever, it seems not improbable that certain fauna such as the Scottish red deer, which are sometimes heavily tick-infested, particularly the younger age groups, may in due course be shown to be infected. The author is not aware of any investigations concerning this possibility, but in view of the importance of this disease in sheep and cattle in certain areas in the United Kingdom, further investigations are warranted.

6. DISEASES DUE TO TRUE NEOPLASMS AND VIRUSES ASSOCIATED WITH TUMOR FORMATION

Because the expectation of life in the majority of species of free-living mammals and birds is comparatively short, true neoplasms are extremely rare. For example, the limited bird-ringing work which has been done in the United Kingdom has shown that the wood pigeon rarely lives for more than four years, and as yet no neoplasm has been discovered in this species. On the contrary, many varieties of neoplasms have been detected in wild animals maintained for long periods in zoological collections, showing that with increasing age, in many instances they are fully susceptible. Neoplasms which have been found in free-living wildlife have involved a variety of hosts, mainly mammals, and the majority of instances are in deer, rabbits and whales.

Tumors of deer

Several varieties of tumors have been recorded in captive, park and free-living deer in Europe and in the United States. In Europe, Runge and Witkowski (1936) encountered a type of adrenal tumor; Stroh (1937) demonstrated a dermoid growth in the eye of a deer; Krause (1938) recorded several cases of adenoma of the liver in roe deer, all the deer being more than five years old ; and Krembs (1939) also mentioned the occurrence of a variety of neoplasms in deer. Later, Bouvier *et al.* (1955) found a glandular epithelioma with metastases in a roe deer. A fibrosarcoma in a wild deer was recorded by Wadsworth (1954) at Vermont in the United States, and Shope (1955) described an infectious fibroma in the same species. In addition to these records, tumor-like skin conditions have been reported from Wisconsin by Chaddock (1939), from Virginia by Quortrup (1946), from California by Herman and Bischoff (1950), and from Canada by Cowan (1951). The growths

described in these reports have been variously called fibromas, papillomas, wart-like structures and fibrosarcomas, but the indications are that probably they were all the same condition. According to Shope, they are also similar to a growth described in New Jersey deer. He has shown, by transmission experiments in susceptible deer, that this condition is definitely an infectious fibroma, and that the causal agent appears to survive for many months in tissue stored in 50 percent glycerol saline at O°F and is filterable through a Berkefeld N filter. This indicates that a virus appears to be responsible for this condition. Kumer (1935) described a papilloma in the chamois in Europe, and it may be that this disease is not dissimilar from the tumors described in deer. Muroma (1954) has also recorded papillomas from six European moose in south Finland. All these skin conditions appear to be closely allied to the wart-like lesions so commonly encountered in domestic cattle in various parts of the world, but there are no records so far of cross-transmission experiments between the different animal species.

Transmissible tumors of wild rabbits

Papillomatosis

Shope and Hurst (1933) showed that warts found in western wild cottontail rabbits were infectious and that the causal agent was undoubtedly a virus. The disease is recognized as occurring mainly in the Midwest United States. The number of warts found on each rabbit varied considerably; from one to several hundreds in severe cases. There was, however, little effect on the general condition of the rabbit. The principal character of the disease appeared to be the presence of horn-like structures leading to the sportsman's term "horned" rabbits. Kidd and Rous (1940) pointed out that in rabbits where the virus has been obtained from another source, variation may occur which may eventually result in malignancy.

Shope fibroma

Shope (1932) found that the cottontail rabbit was subject to a fibrous tumor which was readily transmissible. Subcutaneous tumors, one or several, and loosely attached, were the principal lesions. Microscopically, spindle-celled connective tissue cells were observed but there was no

inflamatory reaction. The disease eventually underwent spontaneous retrogression. This virus is now of considerable importance because of its value as an immunizing agent against myxomatosis.

Myxomatosis

Although myxomatosis could readily be considered in the section of viruses, it is included here because of its close connection to other transmissible diseases characterized by the formation of tumor-like lesions.

In 1898, Sanarelli described the condition, now known as myxomatosis, in laboratory rabbits at Montevideo in Uruguay. He ascribed the disease to a virus, and historically it is interesting to note that it was one of the earliest animal disease viruses to be recognized. Eventually Aragao (1943) showed it to be essentially a mild disease, with little or no mortality, of the wild species in Brazil. Moreover, the common cottontail and jack rabbits of the United States are almost completely resistant; on the contrary, the European rabbit is highly susceptible. Apart from a few isolated instances of infection among hares in France (Jacotot, Vallée and Virat, 1954 a) and rare cases in the United Kingdom and Ireland, no other species appears to be susceptible. It is perhaps worth noting that a skin condition, in some respects similar to myxomatosis, has been known by the writer to have existed in hares in the United Kingdom for many years, but so far the precise cause of this disease has not been determined, although by the distribution of the lesions it closely resembles "orf," a dermatitis of sheep.

The characteristic signs of myxomatosis are inflammation of the periorbital region with a considerable discharge from the eyes and nose, swellings on the head involving the skin of the face and ears, and involvement of the external genitalia. Eventually, swellings may develop over nearly the whole body. Histological examination of an early lesion shows numerous firoblasts and a gelatinous substance which is mainly mucin. Eventually a typical fibroma develops. Experimentally Rivers (1927, 1930) and Findlay (1929) described the microscopic changes in detail, and subsequently Hurst (1937) discussed their findings, contributed some important additional changes, and stated that the histological picture depends on the character of the particular strain of virus employed, as well as on other factors.

A satisfactory immunity can be produced by the inoculation into susceptible rabbits of a vaccine prepared from the Shope fibroma virus, and this is especially useful in those establishments in the United King-

dom, France and elsewhere, where personnel are engaged on veterinary or medical research and where the maintenance of rabbit stocks for such work is imperative.

The literature on myxomatosis has increased considerably, mainly because of the deliberate introduction of the disease to the Australian rabbit population and to the rabbits in France by accidental spread from a deliberately introduced focus on a private estate, quite apart from the more recent spontaneous outbreak in the United Kingdom. From France the disease spread to Belgium, Luxembourg, Germany, the Netherlands and Spain.

Although mosquitoes are the principal vectors of the virus in Australia, initial attempts some years ago to introduce the disease to the rabbits on Skokholm, an island near the Welsh coast of the United Kingdom, failed because of the apparent absence of rabbit fleas (Lockley, 1955). Undoubtedly the rabbit flea plays a most important part in the dissemination of the infection in the United Kingdom. Numerous reports on the disease in Australia have now appeared (Bull and Mules, 1944; Bull, Ratcliffe and Edgar, 1953; Fenner and Marshall, 1954; and Myers *et al.*, 1954). These authors described the general history and epizootiology of the disease in Australia and the present situation, and discussed interesting possibilities, such as migratory wild fowl acting as vectors, when gaps up to 600 miles have occurred between outbreaks.

In France, the recent outbreak has even been described as a national disaster, and in a country where the rabbit is considered as the main sporting quarry for the bulk of the nation's sportsmen, this may well be true. Consequently, numerous articles have now appeared in the French sporting and scientific journals dealing with such important aspects of the disease as epizootiology and immunity (Jacotot, 1953; Jacotot and Vallée, 1953; Jacotot, Vallée and Virat, 1953 a, b; 1954 b, c; 1955 a, b, c, d; and Jacotot *et al.*, 1954).

In the United Kingdom, Thomson (1954) and Lapage (1954) gave general reviews of the situation, and Shanks *et al.* (1955) and Hudson and Mansi (1955) added further information to knowledge of the conditions, the former by describing attempts to introduce the infection to certain islands near the Scottish coast, and the importance of the rabbit flea as a vector, and the last-named authors contributing evidence on the alteration in the character of the virus obtained from surviving rabbits in the United Kingdom after the disastrous epizootic of 1954-55, when much of the rabbit population was destroyed.

The full impact of myxomatosis on the countryside in the United Kingdom, the flora and fauna, sporting and agricultural activities, is not yet fully assessed, and it may be some years before the whole picture is complete; but even now, it is probably true to say that this disease has had a phenomenal effect on the country as a whole, causing great controversy and discussion and changing the way of life of many people. It is a striking example of how a disease of a free-living wild mammal can alter the economy of a country. It has been said, for example, that the virtual disappearance of the rabbit from the United Kingdom has saved agriculture and forestry three or four times the estimated sum of 15 million pounds a year, which was normally expected as an income from the rabbits. Rabbits are, however, at the time of writing, again appearing in the English countryside, and the future of the rabbit population as a whole will soon be decided.

Tumors in whales

It would be difficult to find a better example of a truly free-living wild mammal than the whale in antarctic waters, and it is of considerable interest to note that several tumors have recently been discovered in these animals. Rewell and Willis (1949) had the opportunity, through the courtesy of the " Discovery " Committee, to inspect material collected from the Antarctic Ocean and maintained at the British Museum. They encountered six tumors from a large collection of specimens, namely a uterine fibroma (species of whale not known), a papilloma of the tongue from a blue whale, a mucinous cystadenoma of the ovary from the same species, and three granullosa-cell tumors from a blue whale and two fin whales. The tumors were very small compared with the size of the whales. It is possible that considering the samples available, the granullosa cell tumors may be comparatively common in whales. A further case of ganglioneuroma in a blue whale was recorded by the same authors (1950), and Stolk (1952), working in the Netherlands, described five more cases, collected during 1950-51 by the Dutch whaler " Willem Barendsz. " These consisted of adenoma of the liver in a bottle-nosed dolphin, fibroma of the skin in a fin whale, fibroma of the tongue in the same species, fibroma of the lower jaw in a sperm whale, and a similar tumor in the tongue of the humpback. Cockrill (1960) recorded lipomatous tumors in the fin and blue whales seen during antarctic pelagic

expeditions in 1948-49 and 1951-52. He remarked on their extremely small size and apparently benign character.

Tumors in other wild mammals

Hares in the captive state appear to be subject to neoplasms. For example, Cheatum (1951) described an adenocarcinoma and an epidermoid carcinoma in hares in the United States, but he pointed out that in over 500 autopsies of wild hares of different varieties, no malignant growths had been observed. This agrees with the experience of the author, who has so far never detected a neoplasm in an English hare. On the European continent, however, Zoller (1940) encountered an osteosarcoma in a wild hare in Germany, and Bouvier *et al.* (1954) listed a variety of neoplasms in Swiss hares.

Naturally - occurring fibromas in American wild squirrels, apparently related to Shope's rabbit fibroma, were described by Kilham, Herman and Fisher (1953), and hepatomas in woodchuck and testicular carcinoma in the badger have been found by Habermann, Williams and Eyestone (1954). There is an article on neoplasms in monkeys by Figge (1952), from which it appears probable that monkeys may often be affected with neoplasms, under natural conditions. It is obvious, therefore, that this factor must be borne in mind when using these animals as experimental subjects. In recent years, it has become common practice in some countries to dispose of radioactive waste products in the sea or in inland waterways. The author has already heard of at least one case of osteosarcoma in a muskrat found dying in the vicinity of a lake into which radioactive material had been discharged. When examined, this animal was apparently highly radioactive. Although this may be an isolated case, such a possibility must be borne in mind, especially where the lakes are the habitats of migratory wildfowl.

Tumors in birds

Tumors are rare in birds, although a leiomyoma has been recorded in the caeca of the pheasant, associated with severe *Heterakis* infestation (Krahnert, 1952); and it is probable that such conditions do in fact occur in nature, although rarely noticed. Avian lymphomatosis, produc-

ing a tumor-like condition in the viscera, especially the liver, has been frequently found in game birds in the United Kingdom by the author and by Dr. Clapham (personal communication), the partridge and pheasant being most frequently affected. It appears that this condition in game birds is closely allied to the presence of the disease in neighboring domestic fowl. Jennings (1954) also recorded avian lymphomatosis in three species of wild birds in the United Kingdom, namely, the neural type in a partridge and the visceral type in a shelduck and in a little owl. According to Cowan (1940), many sooty grouse near Cowichan Lake, British Columbia, were observed to be diseased in 1939, and some specimens examined showed tumors affecting the skin of the head; these were subsequently identified as *Epithelioma contagiosum*. Cowan suggested the possibility that they may have been transmitted from bird to bird by biting flies. A similar condition has recently been observed by the authors in British gray partridges, although the precise nature of the tumor has not yet been fully determined.

BIBLIOGRAPHY

Introduction

Elton, C. (1931) *J. Hyg.*, 31, 435.

Diseases due to bacteria

Staphylococcal infection

Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Monographie des maladies du lièvre en Suisse*. Lausanne, Service vétérinaire cantonal et Institut Galli-Valerio, p. 36.
Corsico, G. & Poggi, A. (1953) *Atti Soc. ital. Sci. vet.*, 6, 212.
Jennings, A. R. (1954) *J. comp. Path.*, 64, 356.
McDiarmid, A. (1955) *J. comp. Path.*, 65, 17.
McDiarmid, A. (1944) Unpublished observations.
Smith, H. Williams (1948) *J. comp. Path.*, 58, 179.
Spinelli, A. & Penso, G. (1932) *Clin. vet., Milano*, 55, 173.

Streptococcal infection

Jennings, A. R. (1954) *J. comp. Path.*, 64, 356.
Mantovani, G. & Ceretto, F. (1953) *Atti. Soc. ital. Sci. vet.*, 6, 571.
McDiarmid, A. (1946) Unpublished observations.

Tuberculosis

Beaudette, F. R. & Hudson, C. B. (1929) *J. Amer. vet. med. Ass.*, 74, 1064.
Beaudette, F. R. & Hudson, C. B. (1936) *J. Amer. vet. med. Ass.*, 89, 215.
Beaudette, F. R. & Hudson, C. B. (1942) *J. Amer. vet. med. Ass.*, 101, 274.
Bosworth, T. J. (1940) *J. comp. Path.*, 53, 42.
Bouvier G. (1947) *Schweiz. Arch. Tierheilk.*, 89, 240.
Bouvier G., Burgisser, H. & Schweitzer, R. (1951) *Schweiz. Arch. Tierheilk.*, 93, 689.
Bygyaki, L. (1955) *Bull. agric. Congo belge*, 46, 341.
Christiansen, M. (1931) *Z. InfektKr. Haustiere*, 40, 165.
Christiansen, M., Ottosen, H. E. & Plum, N. (1946) *Skand. VetTidskr.*, 36, 352.
Cowan, I. McT. (1941) *Rep. Provincial Game Commission, Victoria, B. C.*, p. 2.
Cowan, I. McT. (1951) *Proc. 5th Annual Game Convent., Victoria B. C.*, p. 59.

Doyle, T. M. (1943) Personal communication.
Feldman, W. H. (1938) *Avian tuberculosis infections.* London, Baillière, Tindall and Cox.
Grini, O. (1942) Quoted by Grini (1944) *Norsk VetTidsskr.*, 56, 22.
Grini, O. (1944a) *Norsk VetTidsskr.*, 56, 116.
Grini, O. (1944b) *Norsk VetTidsskr.*, 56, 22.
Hadwen, S. (1942) *J. Amer. vet. med. Ass.*, 100, 19.
Hare, T. (1932) *Proc. Roy. S. Med.*, 25, 1500.
Hammond-Smith (1908) *Field*, 18 April.
Harrison, J. M. (1948) *J. Path. Bact.*, 60, 583.
Harrison, J. G. (1955) Personal communication.
Harshfield, G. S., Roderick, L. M. & Hawn, M. C. (1937) *J. Amer. vet. med. Ass.*, 91, 323.
Heidkamp, K. (1939) *Rdsch. Fleischbesch. Trichinensch.*, 40, 103.
Hermansson, K. A. (1943) *Skand. VetTidskr.*, 33, 66.
Hignett, S. L. & MacKenzie, D. A. (1940) *Vet. Rec.* 52, 585.
Hillenbrand, F. (1940) Inaug. Diss. Leipzig., p. 28.
Hulphers, G. & Henricson, T. (1943) *Svensk VetTidskr.*, 48, 245.
Hulphers, G. & Lillengen, K. (1945) *Svensk Jakt* No. 1, 28.
Kleinschmidt, A. & Westphal, W. (1950) *Dtsch. tierärztl. Wschr.*, 57.
De Kock, G. (1938) *S. Afr. med. J.*, 12, 725.
Luke, D. (1954) *Vet. Rec.*, 66, 448.
McDiarmid, A. (1948) *J. comp. Path.*, 58, 128.
McDiarmid, A. (1954) *Field*, 6 May.
Mitchell, C. A. & Duthie, R. C. (1929) *Amer. vet. Rev.*, 19, 134.
Mitchell, C. A. & Duthie, R. C. (1950) *Can. J. comp. Med.*, 14, 109.
Platen, J. (1939) *Berl. Münch. tierärztl. Wschr.*, 55, 536.
Plum, N. (1942) *Skand. VetTidskr.*, 32, 464.
Rac, R. (1951) *Aust. vet. J.*, 27, 209.
Robinson, E. M. (1953) *J. S. Afr. vet. med. Ass.*, 24, 97.
Schmidt, H. W. (1938) *Dtsch. tierärztl. Wschr.*, 46, 482.
Shattock, Seligman, Dudgen & Panton. (1909) *Proc. Roy. Soc. Med.*, 3.
Sinkovic, D. (1954) *Aust. vet. J.*, 30, 215.
Steinhaus, E. A. & Kohls, G. M. (1942) *J. Amer. vet med. Ass.*, 101, 502.
Witte, J. (1940) *Berl. Münch. tierärztl. Wschr.*, 56, 349.

TUBERCULOSIS IN FIELD VOLES

Brooke, W. S. (1941) *Amer. Rev. Tuberc.*, 43, 806.
Griffith, A. S. & Dalling, T. (1940) *J. Hyg.*, 40, 673.
Wagner, J. C., Buchanan, G., Bokkenheusen V. & Leviseur, S. (1958) *Nature*, 181, 284.
Wells, A. Q. (1937) *Lancet*, I, 1221.
Wells, A. Q. (1945) *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 259.
Wells, A. Q. (1953) *J. gen. Microbiol.*, 9, 149.
Wells, A. Q. & Wylie, J. A. H. (1954) *Brit. med. Bull.*, 10, 96.
Wells, A. Q. & Brooke, W. S. (1940) *Brit. J. exp. Path.*, 21, 104.
Young, J. A. & Patterson, J. S. (1949) *J. Hyg.*, 47, 40.

Rat leprosy

Leger, N. (1919) *Bull. Soc. Path. exot.*, 12, 169.
McCoy, G. W. (1913) *Publ. Hlth Bull., Wash.*, No. 61, 27.
Marchoux, E. (1922) *Bull. Acad. Méd., Paris*, 87, 545.
Marchoux, E. & Sorel, F. (1912) *Ann. Inst. Pasteur*, 26, 675, 778.
Ota, M., & Asami, S. (1932) *C. R. Soc. Biol., Paris*, 111, 287.
Stefansky, W. K. (1903) *Zbl. Bakt.*, 33, 481.
Wherry, W. B. (1908) *J. infect. Dis.*, 5, 507.

Salmonellosis, *Escherichia coli* and paracolon infections

Armstrong, W. H. (1942) *Cornell Vet.*, 32, 87.
Block, L., Milzer, A. & Kerdeman, E. (1949) *Gastroenterology*, 12, 508.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1955) *Schweiz. Arch. Tierheilk.*, 97, 324.
Bruner, D. V. & Moran, A. B. (1949) *Cornell Vet.*, 39, 53.
Buxton, A. & Field, H. I. (1949) *Rep. 14th Int. vet. Congress, Lond.*, 2, 278.
Cass, J. S. & Williams, J. E. (1947) *J. Amer. vet. med. Ass.*, 111, 282.
Chitty, D. & Southern, H. N. (1954) *Control of rats and mice*. London, Oxford University Press, 3, 40.
Cronk, P. G. (1952) *Lancet*, 263, 541.
Danysz, J. (1900) *Ann. Inst. Pasteur*, 14, 193.
Dornall, C. R. (1948) *Gastroenterology*, 10, 366.
Edwards, P. R. & Bruner, D. W. (1941) *Proc. Soc. exp. Biol., N. Y.*, 48, 240.
Fitzgerald, B. Vesey. (1946) *British game*. London, Collins, p. 65.
Graham, R. (1936) *J. Amer. vet. med. Ass.*, 88, 763.
Harbourne, J. F. (1955) *J. comp. Path.*, 65, 250.
Hinshaw, W. R. & McNeil, E. (1946a) *J. Bact.*, 51, 281.
Hinshaw, W. R. & McNeil, E. (1946b) *J. Bact.*, 51, 397.
Hülphers, G. & Henricson, T. (1943) *Svensk VetTidskr.*, 48, 245.
Jennings, A. R. (1954) *J. comp. Path.*, 64, 356.
Jones, E. R. & Wright, H. D. (1936) *Lancet*, 1, 22.
Kernohan, R. J. (1952) *Lancet*, 262, 1090.
Kirkpatrick, C. M., Moses, H. E. & Baldini, J. T. (1952) *Amer. J. vet. Res.*, 13, 99.
Khalil, A. M. (1938) *J. Hyg.*, 38, 75.
Kumerloeve, H. & Steiniger, F. (1952) *Dtsch. tierärztl. Wschr.*, 59, 312.
Lee, P. E. & MacKerras, I. M. (1955) *Aust. J. exp. Biol. med. Sci.*, 33, 117.
McDiarmid, A. (1953) *Vet. Rec.*, 66, 460.
Oppenheimer, C. H. & Kelly, A. L. (1952) *Science*, 115, 527.
Savage, W. G. & White, P. B. (1923) *J. Hyg.*, 21, 258.
Spray, R. S. (1926) *J. Amer. med. Ass.*, 86, 109.
Ugorski, L. (1952) *Méd. vét., Varsovie*, 8, 540.
Van Dorssen, C. A. (1935) *Tijdschr. Diergeneesk.*, 62, 1263.
Van Dorssen, C. A. (1953) *Tijdschr. Diergeneesk.*, 78, 161.
Varela, G., Olarte, J. & Mata, F. (1948) *Rev. Inst. Salubr. Enferm. trop.*, 9, 239.
Watts, P. S. (1951) *13th Rep. Inst. Med. & Vet. Sci. S. Australia, Vet. Path. Section.*
Willführ & Wendtlandt (1921) *Z. Hyg. Infektkr.*, 94, 192.

PLAGUE (*Pasteurella pestis*)

Davis, D. H. S. (1953) *J. Hyg.*, 51, 427.
Eastwood, A. & Griffith, F. (1914) *J. Hyg.*, 14, 285.
Holdenried, R. & Morlan, H. B. (1955) *J. infect. Dis.*, 96, 133.
Jellison, W. L. (1939) *Publ. Hlth Rep., Wash.*, 54, 792.
McAlister, G. H. & Brooks, R. St. J. (1914) *J. Hyg.*, 14, 316.
McCoy, G. W. (1910) *J. Hyg.*, 10, 589.
McCoy, G. W. (1911) *Publ. Hlth Bull., Wash.*, No. 43.
McCoy, G. W. & Wherry, W. B. (1909) *J. infect. Dis.*, 6, 670.
Meyer, K. F. *et al.* (1943) *J. infect. Dis.*, 73, 144.
Mitchell, J. A. (1921) *J. Hyg.*, 20, 377.
Mitchell, J. A. (1930) *J. Hyg.*, 39, 394.
Petrie, G. F. & McAlister, G. H. (1911) *Rep. Med. Off. Loc. Govt. Ed.* 1910-11, p. 91.
Pirie, J. H. H. (1929) *Publ. S. Afr. Inst. med. Res.*, 4, 218.
Plague Research Commission (1907) *J. Hyg.*, 7, 323, 693.
Williams, C. L. & Kemmerer, T. W. (1923) *Publ. Hlth Rep., Wash.*, 38, 1873.

OTHER CONDITIONS ASSOCIATED WITH *Pasteurella*

Bivins, J. A. (1953) *Cornell. Vet.*, 43, 241.
Bivins, J. A. (1955) *Cornell Vet.*, 45, 180.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1953) *Schweiz. Arch. Tierheilk.*, 95, 626.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1952) *Schweiz. Arch. Tierheilk.*, 94, 475.
Cowan, I. McT. (1951) *Rep. 5th Annual Game Convent., Victoria, B. C.*, p. 62.
Hülphers, G. & Henricson, T. (1943) *Svensk. VetTidskr.*, 48, 245.
Hudson, C. B. (1944) *J. Amer. vet. med. Ass.*, 104, 211.
Jennings, A. R. (1954) *J. comp. Path.*, 64, 356.
Kaschula, V. R. & Truter, D. E. (1951) *J. S. Afr. vet. med. Ass.*, 22, 191.
Raggi, L. G. & Stratton, G. S. (1954) *Cornell Vet.*, 44, 229.
Muroma, E. (1951) *Suomen Riista*, 6, 159.
Quortrup, E. R., Queen, F. B. & Merovka, L. J. (1946) *J. Amer. vet. med. Ass.*, 108, 94.
Suarez, J. G. & Ilazabal, L. L. (1941) *Rev. Med. vet. B. Aires*, 23, 145.

PSEUDOTUBERCULOSIS (*Pasteurella pseudotuberculosis*)

Beaudette, F. R. (1940) *J. Amer. vet. med. Ass.*, 97, 151.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Monographie des maladies du lièvre en Suisse.* Lausanne, Service vétérinaire cantonal et Institut Galli-Valerio, p. 41.
Christiansen, M. (1939) *Dansk. Jagt.* 1.
Christiansen, M. (1949) *Dansk orn. Foren. Tidsskr.*, 43, 189.
Clapham, P. (1953) *Nature*, 172, 353.
Jennings, A. R. (1954) *J. comp. Path.*, 64, 356.

Muroma, E. (1951) *Suomen Riista*, 6, 159.
Olt, A. (1937) *Z. Infektkr. Haustiere*, 52, 89.
Pullar, E. M. (1932) *Aust. vet. J.*, 8, 181.
Thal, E. (1953) *Proc. 15th Int. vet. Congress*, 1, 67.
Zibert, S. (1939) *Jugoslov. vet. Glasn.*, 19, 174.

PSEUDOTUBERCULOSIS (*Corynebacterium*)

Cowan, I. McT. (1951) *Proc. 5th Annual Game Convent., Victoria B. C.*, p. 61.
Hammersland, H. & Joneschild, E. M. (1937) *J. Amer. vet. med. Ass.*, 91, 186.
Schoop, G. (1940) *Dtsch. tierärztl. Wschr.*, 47, 253.
Scone (1927) *Brit. Birds*, 21, 19.
Seghetti, L. & McKenny, F. D. (1941) *J. Amer. vet. med. Ass.*, 98, 129.

BRUCELLOSIS

Agababyan, M. M. (1940) *Veterinariya, Moskva*, No. 4, 53.
Bendtsen, H., Christiansen, M. & Thomsen, A. (1954) *Nord. VetMed.*, 6, 11.
Bosworth, T. J. (1937) *J. comp. Path.*, 50, 345.
Buddle, M. B. & Boyes, B. W. (1953) *Aust. vet. J.*, 29, 145.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Schweiz. Arch. Tierheilk.*, 96, 85.
Burgisser, H. G. (1949) *Schweiz. Arch. Tierheilk.*, 91, 273.
Burgisser, H. G. (1951) *Schweiz. Arch. Tierheilk.*, 93, 299.
Burgisser, H. G. (1952) *Schweiz. Arch. Tierheilk.*, 94, 554.
Burgisser, H. G. (1954) *Rev. Path. gén., Paris*, 54, 654.
Jacotot, H. & Vallée, A. (1951a) *Bull. Acad. nat. Méd.*, 135, 430.
Jacotot, H. & Vallée, A. (1951b) *Ann. Inst. Pasteur*, 80, 214.
Karadinovsky, J. A. (1936) *C. R. Soc. Biol., Paris*, 121, 1611.
Katz, J. S. (1941) *J. Amer. vet. med. Ass.*, 99, 24.
Kolesnik, N. G. (1941) *Uchen. zap. Kazan. Vet. Inst.*, 53, 168.
Lee, A. M. & Turner, M. E. (1937) *J. Amer. vet. med. Ass.*, 90, 637.
McDiarmid, A. (1951) *Vet. Rec.*, 63, 469.
Manzullo, A. (1935) *Rev. Zootech.*, 22, 133.
Menton, J. (1937) *Brit. med. J.*, Dec. 25, 1273.
Moore, T. (1947) *Canad. J. comp. Med.*, 11, 131.
Preun, B. (1938) *Dtsch. tierärztl. Wschr.*, 46, 804.
Roux, L. & Bouvier, G. (1946) *Schweiz. Arch. Tierheilk.*, 88, 507.
Schiel, O. (1936) *Z. Fleisch- u. Milchhyg.*, 47, 114.
Schmid, G. & Klinger, K. (1953) *Proc. XV Int. vet. Congress*, Part I, 1, 85.
Selmi, G. (1941) *Riv. ital. Igiene*, 1, 1083.
Thomsen, A. (1959) *Nord. VetMed.*, 11, 709.
Witte, J. (1941) *Berl. Münch. tierärztl. Wschr.*, 57, 128.

TULAREMIA

Bouvier, G., Burgisser, H. & Schneider, P. A. (1951) *Schweiz. Arch. Tierheilk.*, 93, 821.

Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Monographie des maladies du lièvre en Suisse.* Lausanne, Service vétérinaire cantonal et Institut Galli-Valerio.
Foote, H. B. *et al.* (1943) *J. Amer. Wat. Wks Ass.*, 35, 902.
Francis, E. (1919) *Publ. Hlth Rep., Wash.*, 34, 2061.
Francis, E. (1923) *Publ. Hlth Rep., Wash.*, 38, 1391.
Hammersland, H. L. & Joneschild, E. M. (1940) *J. Amer. vet. med. Ass.*, 96, 96.
Jellison, W. L. *et al.* (1950) *Publ. Hlth Rep., Wash.*, 65, 1219.
Jellison, W. L. *et al.* (1942) *Amer. J. Hyg.*, 36, 168.
Karpoff, S. P. & Antonoff, N. I. (1936) *J. Bact.*, 32, 243.
Kohls, G. M. & Steinhaus, E. A. (1943) *Publ. Hlth Rep., Wash.*, 58, 842.
Langford, E. V. (1954) *Canad. J. comp. Med.*, 18, 28.
McCoy, G. W. (1911) *Publ. Hlth Bull., Wash.*, No. 43, 53.
McCoy, G. W. & Chapin, C. W. (1912) *J. infect. Dis.*, 10, 61.
Miller, A. A. (1935) *Sovetskaia Urachebuais. Gazeta, Leningr.*, 39, 187.
Olin, G. (1938) *Hygiea, Stockh.*, 100, 236.
Parker, R. R., Spencer, R. R. & Francis, E. (1924) *Publ. Hlth Rep., Wash.*, 39, 1057.
Parker, R. R. *et al.* (1951) U. S. National Institute of Health Bulletin No. 193.
Roubakine, A. (1930) *Mon. epidem. Rep. Hlth Sect. L. o. N.*, R. E. No. 134.
Schmidt, B. (1947) *Z. Hyg. InfektKr.*, 127, 139.
Scott, J. W. (1940) *Science*, 91, 263.
Seton, E. T. (1929) *Lives of game animals.* New York, Doubleday, Doran, 4, 489.
Sinai, G. I. & Voskresensky, B. V. (1943) *In* Khatenever, L. M., ed. *Tularemia infections.* Moscow, All-Union Institute of Experimental Medicine.
Thjotta, T. (1930) *Bull. Hyg., Lond.*, 5, 490.
Thjotta, T. (1931a) *Bull. Hyg., Lond.*, 6, 355.
Thjotta, T. (1931b) *J. infect. Dis.*, 49, 99.
Tumansky, V. & Kolesnikova, Z. (1935) *Rev. Microbiol., Saratov*, 14, 269.
Voskresensky, B. V. (1943) *In* Khatenever, L. M., ed. *Tularemia infections.* Moscow, All-Union Institute of Experimental Medicine.

Botulism

Coburn, D. R. & Quortrup, E. R. (1939) *Trans. 4th N. Amer. Wild Life Conf., Washington*, 359.
Kalmbach, E. R. & Gunderson, M. F. (1934) *U. S. Dep. Agric. Tech. Bull.*, 411, 81.
Kalmbach, E. R. (1935) *J. Amer. vet. med. Ass.*, 87, 183.
Kalmbach, E. R. (1939) *J. Amer. vet. med. Ass.*, 94, 187.
Pyle, N. J. & Brown, R. M. (1939) *J. Amer. vet. med. Ass.*, 94, 436.
Gunnison, J. B. & Coleman, G. E. (1932) *J. infect. Dis.*, 51, 542.
Quortrup, E. R. & Shillinger, J. E. (1941) *J. Amer. vet. med. Ass.*, 99, 382.
Quortrup, E. R. & Sudheimer, R. L. (1942) *Trans. 7th N. Amer. Wild Life Conf.*, 284.
Wetmore, A. (1918) *U. S. Dep. Agric. Bull.* 672, 26.

Other clostridial infections

Green, R. G. & Larson, C. (1938) *Amer. J. Hyg.*, 28, 190.
McKenney, F. D. (1938) *Trans. 3rd N. Amer. Wild Life Conf.*, 886.
Taylor, A. W. & Gordon, W. S. (1940) *J. Path. Bact.*, 50, 271.

Anthrax

Shrewsbury, J. F. D. & Barson G. J. (1952) *J. Path. Bact.*, 64, 605.
Thomas, A. D. & Neitz, W. O. (1933) *S. Afr. J. Sci.*, 30, 419.

Actinomycosis and related diseases

Bergey, D. H. (1939) *Manual of determinative bacteriology.* 5th ed. London, Baillière, Tindall and Cox, p. 873.
Bosse (1938) *Tierärztl. Rdsch.*, 44, 641.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1952) *Schweiz. Arch. Tierheilk.*, 94, 478.
Cowan, I. McT. (1951) *Proc. 5th Annual Game Convent., Victoria B. C.*
McDiarmid, A. & Austwick, P. K. C. (1954) *Nature*, 174, 843.
Munch-Petersen, E. (1954) *Aust. vet. J.*, 30, 297.
Ryff, J. F. (1953) *J. Amer. vet. med. Ass.*, 122, 78.
Waterson, A. P. & Wedgwood, J. (1953) *Lancet*, 264, 472.

Listeriosis

Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Monographie des maladies du lièvre en Suisse.* Lausanne, Service vétérinaire cantonal et Institut Galli-Valerio.
Henricson, T. (1943) *Svensk. VetTidskr.*, 48, 1.
Lillengen, K. (1942) Abstract in *Zbl. Bakt.*, 1 (Ref.) 142, 494.
Pirie, H. (1927) *Publ. S. Afr. Inst. med. Res.*, 3, 163.
Vallée, A. (1952) *Ann. Inst. Pasteur*, 83, 832.

Erysipelas (*Erysipelothrix rhusiopathiae*)

Bourgeois, E. (1944) *Schweiz. Arch. Tierheilk.*, 86, 32.
Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Monographie des maladies du lièvre en Suisse.* Lausanne, Service vétérinaire cantonal et Institut Galli-Valerio, p. 62.
Connell, R. (1954) *Canad. J. comp. Med.*, 18, 22.
Christiansen, M. (1949) *Dansk orn. Foren. Tidsskr.*, 43, 189.
Hülphers, G. & Henricson, T. (1943) *Svensk VetTidskr.*, 48, 245.
Lebeda, K. (1940) *Z. InfektKr. Haustiere*, 56, 229.
Stiles, G. W. (1944) *Amer. J. vet. Res.*, 5, 243.
Taylor, A. W. (1954) Personal communication.
Vianello, G. (1938) *Clin. vet., Milano*, 61, 234.
Wayson, N. E. (1927) *Publ. Hlth Rep., Wash.*, 42, 1489.
Wellmann, G. & Leibke, H. (1960) *Berl. Munch. tierärztl. Wschr.*, 73, 329.

Necrobacillosis

Lord, G. (1953) *Proc. 89th Annual Congress Amer. vet. med. Ass.*, 546.

Bartonella infection of moles

Graham-Smith, G. S. (1905) *J. Hyg.*, 5, 453.
Weinman, D. (1944) *Trans. Amer. phil. Soc.*, 33, pt. III, 243.

Leptospirosis (*Leptospira icterohaemorrhagiae*)

Balfour, A. (1922) *Parasitology*, 14, 282.
Bessemans, A. & Thiry, U. (1929) *C. R. Soc. Biol., Paris*, 101, 486.
Broom, J. C. & Gibson, E. A. (1953) *J. Hyg.*, 51, 416.
Buchanan, G. (1927) *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 113.
Cameron, G. C. & Irwin, D. A. (1929) *Canad. publ. Hlth J.*, 20, 386.
Dunkin, G. W. (1926) *Vet. J.*, 82, 147.
Ido, Y. *et al.* (1917) *J. exp. Med.*, 26, 341.
Middleton, A. D. (1929) *J. Hyg.*, 29, 219.
Schuffner, W. (1934) *Trans. R. Soc. trop. Med. Hyg.*, 28, 7.
Ssinjuschina, M. N. (1929) *Zbl. Bakt.*, 114, 199.
Stevenson, A. C. (1922) *Amer. J. trop. Med.*, 2, 77.
Uhlenhuth & Zuelzer (1921) *Zbl. Bakt.*, 85, Beiheft, p. 141.

Other infections with *Leptospira* spp.

Babudieri, B. (1953) *Advances in the control of zoonoses.* FAO Agric. Studies No. 25, p. 117.
Broom, J. C. & Coghlan, J. D. (1958) *Lancet*, ii, 1041.
Broom, J. C. & Coghlan, J. D. (1960) *Lancet*, June 18, 1326.
Ido, Y., Ito, H. & Wani, H. (1918) *J. exp. Med.*, 28, 435.
Latyshev, N. I. & Pozgvaj, T. T. (1936) *In* Patogennye zhivotnye. *Trudy Otd. Parazitol. Vsesoiuz. Inst. Eksper. Med. Gorkogo*, 2, 79.
Mino, P. (1942) *Klin. Wschr.*, 21, 337.
Parnas, J. *et al.* (1961) *J. infect. Dis.*, 108, 243.
Trainer, D. O. jr., Karstad, L. & Hanson, R. P. (1961) *J. infect. Dis.*, 108, 278.
Van der Hoeden, J. (1955) *J. Amer. vet. med. Ass.*, 126, 207.

Rat bite fever

Futaki, K. *et al.* (1917) *J. exp. Med.*, 25, 33.
Robertson, A. (1924) *Ann. trop. med. Parasit.*, 18, 157.
Schockaert, J. (1928), *C. R. Soc. Biol., Paris*, 98, 595.

Relapsing fever

Bruce, D. *et al.* (1911) *Rep. Sleeping Sickness Commission, Lond.*, 11, 184.
Carpano, M. (1913) *Ann. Igiene (sper.)*, 23, 215.
Leger, A. (1917) *Bull. Soc. Path. exot.*, 10, 280.
Lingard, A. (1907) *J. trop. vet. Sci.*, 2, 261.
Nicolle, C., Anderson, C. & Colas-Belcour, J. (1927) *C. R. Acad. Sci., Paris*, 185, 334.

Todd, J. L. & Wolbach, S. B. (1912) *J. med. Res.*, 26, 195.
Vianna, G. de Figueiredo & Cruz, B. G. (1912) *Brazil-med.*, 26, 912.

Avian Spirochetosis

Fantham, H. B. (1910) *Proc. zool. Soc. Lond.*, 692.
Franchini, G. (1924) *Ann. Inst. Pasteur*, 38, 470.

Rabbit syphilis

Bayon, H. (1913) *Brit. med. J.*, *ii*, 1159.
Bessemans, A. (1928) *C. R. Soc. Biol., Paris*, 99, 331.
Bessemans, A. & Geest, E. de (1928) *C. R. Soc. Biol., Paris*, 99, 334.
Ross, E. H. (1912) *Brit. med. J.*, *ii*, 1653.

Diseases due to fungi

Aspergillosis

Cowan, I.McT. (1943) *Murrelet*, 24, 29.
Davis, W. A. & McClung, L. S. (1940) *J. Bact.*, 40, 321.
Herman, C. M. & Rosen, M. N. (1947) *Condor*, 49, 212.
Hülphers, G., Lillengen, K. & Henricson, T. (1941) *Svensk Jakt*, 6, 250.
Landy, L. (1937) Thesis, Budapest, p. 14.
McDiarmid, A. (1952) *Field*, 1028.
McDiarmid, A. (1955) *J. comp. Path.*, 65, 246.
Motton, S. J. (1945) Personal communication.
Rothschild, M. & Clay, T. (1952) *Fleas, flukes and cuckoos.* London, Collins, p. 243.
Surroy Dane, D. (1948) *J. Anim. Ecol.*, 17, 158.
Sörum, L. (1950) *Fugleviltundersökelser pa laboratoriet N. J. F. F.* 55.
Zeliff, G. C. (1943) *Bird banding*, 14, 127.

Coccidiodomycosis

Ashburn, L. L. & Emmons, C. W. (1942a) *Amer. J. Path.*, 18, 753.
Ashburn, L. L. & Emmons, C. W. (1942b) *Arch. Path.*, 34, 791.
Dickson (1938) *J. Amer. med. Ass.*, 111, 1362.
Emmons, C. W. (1943a) *J. Bact.*, 45, 306.
Emmons, C. W. (1943b) *Publ. Hlth Rep., Wash.*, 58, 1.

Haplomycosis (adiospiromycosis)

Carmichael, J. W. (1951) *Mycologia*, 43, 605.
Dowding, E. S. (1947a) *Mycologia*, 39, 372.
Dowding, E. S. (1947b) *Canad. J. Res.*, E. 25, 193.
Dowding, E. S. (1948) *Canad. J. Res.*, E. 26, 265.
Elton, C. (1931) *J. Hyg.*, 31, 435.

Emmons, C. W. (1948) *Proc. 4th Int. Congress Trop. Med. and Malaria*, 2, 1278.
Emmons, C. W. & Ashburn, L. L. (1942) *Publ. Hlth Rep., Wash.*, 57, 1715.
Emmons, C. W. & Jellison, W. L. (1960) *Ann. N. Y. Acad. Sci.*, 89, 91.
Erickson, A. B. (1949) *J. Wildlife Mgmt*, 13, 419.
Jellison, W. L. (1947) *Proc. helminth. Soc. Wash.*, 14, 75.
Jellison, W. L. (1950) *Publ. Hlth. Rep., Wash.*, 65, 1057.
Jellison, W. L. (1954) *Publ. Hlth Rep., Wash.*, 69, 996.
Jellison, W. L., Helminen, M. & Vinson, J. W. (1960) *Ann. Med. Exper. Fenn.*, 38, 3.
McDiarmid, A. & Austwick, P. K. C. (1954) *Nature*, 174, 843.
Menges, R. W. & Habermann, R. T. (1954) *Amer. J. Hyg.*, 60, 106.

Histoplasmosis

Anon (1952) *Lancet*, 262, 1148.
Emmons C. W. & Ashburn, L. L. (1948) *Publ. Hlth Rep., Wash.*, 63, 1416.
Emmons, C. W., Bell, J. A. & Olsen, B. J. (1947) *Publ. Hlth Rep., Wash.*, 62, 1642.
Menges, R. W., Furcolow, M. L. & Hinton, A. (1954) *Amer. J. Hyg.*, 59, 113.

Sporotrichosis

Ainsworth, G. C. (1954) *Vet. Rec.*, 66, 844.
Hagan, W. A. (1943) *The infectious diseases of domestic animals.* New York, Comstock.

Ringworm (dermatophytes)

Ainsworth, G. C. (1954) *Vet. Rec.*, 66, 844.
Blanc, G. & Catanei, A. (1944) *Arch. Inst. Pasteur Algér.*, 22, 157.
Charles, V. K. (1940) *J. Wash. Acad. Sci.*, 30, 338.
Connor, J. J. (1932) *Med. J. Aust.*, 19, 765.
Cook, M. D. & Graham, R. (1936) *J. Amer. vet. med. Ass.*, 89, 321.
Delamater, E. D. (1939) *Mycologia*, 31, 519.
Du Bois (1929) *Ann. Derm., Paris.*, 10, 1359.
Eddoes, A. (1898) *Trans. derm. Soc. G. B. I.*, 4, 32.
Hasegawa, M. T. & Yamamoto, K. (1936) *Jap. J. Derm. Urol.*, 39, 23.
Mantovani, G. & Ceretto, F. (1953) *Atti. Soc. ital. Sci. vet.*, 6, 571.
Pätiälä, R. & Härö, S. (1950) *Rev. Fungi, Karstenia*, I.
Poland, M. K. (1938) *Ned. Tjdschr. Geneesk.*, 82, (II), 2114.
Shaw, F. W. & Wampler, F. J. (1933) *Virginia med. Mon.*, 59, 742.
Vilanova, X. & Casanovase, M. (1951) *Pr. méd.*, 59, 1760.

Diseases due to viruses

Foot-and-mouth disease

Bartels & Claassen, P. (1936a) *Berl. Münch. tierärztl. Wschr.*, 52, 230.
Bartels & Claassen, P. (1936b) *Berl. Münch. tierarztl. Wschr.*, 52, 455.
Bullough, W. S. (1942) *Proc. roy. Soc.*, (B) 131, 1.

Campion, R. L. (1950) *Gac. vet., B. Aires*, 12, 3.
Christiansen, M. (1939) *Maanedsskr. Dyrlaeg.*, 51, 385.
Cohrs, P. & Weber-Springe, W., (1939) *Dtsch. tierärztl. Wschr.*, 47, 97.
Eccles, A. (1939) *Bull. int. Off. Epiz.*, 18, 118.
Elton, C. (1937) *5th Rep. Foot-and-Mouth Disease Committee, Lond.*, p. 379.
Haq, M. M. (1951) *Proc. 3rd Pakistan Sci. Conf., Dacca*, Pt. III, p. 89.
Longley, E. O. (1937) *5th Rep. Foot-and-Mouth Disease Committee, Lond.*, p. 56.
Magnusson, H. (1939) *Dtsch. tierärztl. Wschr.*, 47, 509.
McLauchlan, J. D. & Henderson, W. M. (1947) *J. Hyg.*, 45, 474.
Mettam, A. E. (1914) *Rep. 10th Int. vet. Congress*, 2, 105.
Sallinger (1939) *Berl. Münch. tierärztl. Wschr.*, Feb. 10, 89.
Wilson, W. W. & Matheson, R. C. (1952) *Agriculture, Lond.*, 59, 213.

Vesicular stomatitis

Karstad, L. H. *et al.* (1956) *J. Amer. vet. med. Ass.*, 129, 95.
Karstad, L. H. & Hanson, R. P. (1957). *Amer. J. vet. Res.*, 18, 162.

Rift valley fever

Rivers, T. M. ed. (1952) *Viral and rickettsial infections of man.* 2nd ed. London, Lippincott.
Smithburn, K. C., Haddow, A. J. & Gillett, J. D. (1948) *Brit. J. exp. Path.*, 29, 107.
Smithburn, K. C., Haddow, A. J. & Lumsden (1949) *Brit. J. exp. Path.*, 30, 35.

Rabies

Anon. (1954) *Canad. J. comp. Med.*, 18, 313.
Ballantyne, E. E. & O'Donoghue, J. G. (1954) *J. Amer. vet. med. Ass.*, 125, 316.
Burton, H. W. (1950) *J. Bombay nat. Hist. Soc.*, 49, 528.
Chitty, H. (1950) *J. Anim. Ecol.*, 19, 180.
Courter, R. D. (1954) *Vet. Med.*, 49, 206.
Cowan, I. McT. (1949) *J. Mammal.*, 30, 396.
Enright, J. B. *et al.* (1955) *Proc. Soc. exp. Biol., N. Y.*, 89, 94.
Fagan, R. (1953) *Proc. 89th Annual Congress A. V. M. A.*, 1952, 394.
Hirleman, W. B. (1942) *N. Amer. Vet.*, 23, 193.
Herrenberger, R. (1952) *Arch. Inst. Pasteur Algér.*, 30, 371.
Kodrnja, E. (1952) *Veterinaria, Sarajevo*, 1, 687.
Monila, A. (1938) *Bol. Soc. brasil. Med. vet.*, 8, 103.
Pandit, S. R. (1951) *Indian med. Gaz.*, 85, 441.
Pawan, J. L. (1936) *Ann. trop. Med. Parasit.*, 30, 401.
Pawan, J. L. (1948) *Ann. trop. Med. Parasit.*, 42, 173.
Plummer, P. J. G. (1954) *Bull. World Hlth Org.*, 10, 767.
Pritchett, H. D. (1938) *J. Amer. vet. med. Ass.*, 92, 563.
Pullar, E. M. & McIntosh, D. S. (1954) *Aust. vet. J.*, 30, 326.
Schoop, G. (1950) *Mh. Prakt. Tierheilk.*, 2, 65.
Tice, F. J. & Evans, W. M. (1942) *Cornell Vet.*, 32, 98.
Young, K. S. (1953) *Proc. 89th Annual Congress, A. V. M. A.*, 1952, 416.
Verteuil, E. de & Urich, F. W. (1936) *Trans. R. Soc. trop. Med. Hyg.*, 29, 317.

PSEUDORABIES (AUJESZKY'S DISEASE)

Shope, R. E. (1934) *Science*, 80, 102.
Shope, R. E. (1935) *J. exp. Med.*, 62, 101.

PSITTACOSIS

Anon. (1939) *Lancet*, 236, 708.
Burnet, F. M. (1935) *J. Hyg.*, 35, 412.
Burnet, F. M. (1939) *Med. J. Aust.*, 1, 545.
Haagen, E. & Mauer, G. (1938) *Zbl. Bakt.*, 143, 81.
Miles, J. A. R. & Shrivastav, J. B. (1951) *J. Anim. Ecol.*, 20, 195.
Mykytowycz R., Surrey Dane, D. & Beech, M. (1955) *Aust. J. exp. Biol. med. Sci.*, 33, 629.
Pinkerton, H. & Swank, R. L. (1940) *Proc. Soc. exp. Biol., N. Y.*, 45, 704.
Rasmussen, R. K. (1938) *Ugeskr. Laeg.*, 100, 989.
Rubin, H. *et al.* (1951) *Proc. Soc. exp. Biol., N. Y.*, 78, 696.
Rubin, H. (1954) *J. infect. Dis.*, 94, 1.

OPOSSUM DISEASE

Roca-Garcia, M. (1949) *J. infect. Dis.*, 85, 275.

POX DISEASES IN BIRDS

Dobson, N. (1937) *J. comp. Path.*, 50, 401.
French, E. L. & Reeves, W. C. (1954) *J. Hyg.*, 52, 551.
Geurden, L. (1941) *Vlaams Diergeneesk. Tijdschr.*, 10.
Holmes, F. C. (1948) The filterable viruses, supplement No. 2. *In* Bergey, D. H. *Manual of determinative bacteriology*. 6th ed. London, Baillière, Tindall and Cox.
Kossack, C. W. & Hanson, H. C. (1954) *J. Amer. vet. med. Ass.*, 124, 199.
McGaughey, C. A. & Burnet, F. M. (1945) *J. comp. Path.*, 55, 201.
Shattock, S. G. (1898) *Trans. path. Soc. Lond.*, 49, 394.
Syverton, J. T. & Cowan, I. McT. (1944) *Amer. J. vet. Res.*, 5, 215.

CONTAGIOUS ECTHYMA OF SHEEP

Conell, R. (1954) *Canad. J. Comp. Med.*, 18, 59.

FOWL PLAGUE (TRUE FOWL PEST)

Hagan, W. A. (1943) *The infectious diseases of domestic animals.* New York, Comstock, p. 628.

PUFFINOSIS

Miles, J. A. R. & Stoker, M. G. P. (1948) *Nature*, 161, 1016.
Stoker, M. G. P. & Miles, J. A. R. (1953) *J. Hyg.*, 51, 195.
Surrey Dane, D. (1948) *J. Anim. Ecol.*, 17, 158.
Surrey Dane, D., Miles, J. A. R. & Stoker, M. G. P. (1953) *J. Anim. Ecol.*, 22, 123.

NEWCASTLE DISEASE (PSEUDO-FOWL PEST). ALSO REFERRED TO IN THE UNITED KINGDOM AS FOWL PEST

Blaxland, J. D. (1951) *Vet. Rec.*, 63, 731.
Gillespie, J. H., Kessel, B. & Fabricant, J. (1950) *Cornell Vet.*, 40, 93.
Gustafson, D. P. & Moses, H. E. (1953a) *Amer. J. vet. Res.*, 14, 581.
Gustafson, D. P. & Moses, H. E. (1953b) *Proc. 89th Annual Conference A. V. M. A.*, 1952, 281.
Mantovani, G. & Ceretto, F. (1953) *Atti. Soc. ital. Sci. vet.*, 6, 571.
Parnaik, D. T. & Dixit, S. G. (1953) *Indian vet. J.*, 30, 145.
Popovič, B. (1951) *Acta Vet., Belgrade*, 1, 168.
Wilson, J. E. (1950) *Vet. Rec.*, 62, 33.
Zuydam, D. M. (1951) *Tijdschr. Diergeneesk.*, 76, 237.

OTHER VIRUS INFECTIONS IN BIRDS

Olson, N. O. (1950) *Proc. 54th Annual Meeting U. S. Live Stock Sanitary Association*, 171.
Todd, C. (1930) *A system of bacteriology in relation to medicine.* London, Medical Research Council, 7, 231.

CANINE DISTEMPER

Hagan, W. A. (1943) *The infectious diseases of domestic animals.* New York, Comstock, p. 576.
Helmboldt, C. F. & Jungherr, E. L. (1955) *Amer. J. vet. Res.*, 16, 463.
Robinson, V. B., Newberne, J. W. & Brooks, D. M. (1955). *J. Amer. vet. med. Ass.*, 131, 276.

SWINE FEVER (HOG CHOLERA)

De Tray, D. E. (1957) *J. Amer. vet. med. Ass.*, 130, 537.
Englert, H. K. (1953) *Tierärztl. Umsch.*, 8, 124.
Hutter, K. (1953) *Mh. VetMed.*, 8, 109.
Saburov, A. N. & Sokk, N. V. (1937) *Sovyet. Vet.* No. 6, 45.

RINDERPEST

Carmichael, J. (1938) *J. comp. Path.*, 51, 264.
Guyaux, R. (1951) *Bull. agric. Congo belge*, 42, 123.

AFRICAN HORSE SICKNESS

Hagan, W. A. (1943) *The infectious diseases of domestic animals.* New York, Comstock.

MALIGNANT CARARRHAL FEVER

Mettam, R. W. M. (1936) *Conf. vet. Res., Brit. E. Afr.*, p. 108.

YELLOW FEVER

Balfour, A. (1914) *Lancet*, 1, 1176.
Dick, G. W. A. (1953) *Brit. med. Bull.*, 9, 215.
Soper, F. L. (1936a) *Quart. Bull. Hlth Org. L. o. N.*, 5, 19.
Soper, F. L. (1936b) *Rev. Hyg. Rio de J.*, 10, 107.

EQUINE ENCEPHALOMYELITIS

Beaudette, F. R. (1951) *Vet. Ext. Quart. Univ. Pa.*, 51, 96.
Beaudette, F. R. *et al.* (1952) *J. Amer. vet. med. Ass.*, 121, 478.
Burnet, F. McF. (1953) *Publ. Hlth Rep., Wash.*, 68, 102.
Cox, H. R., Jellison, W. L. & Hughes, L. E. (1941) *Publ. Hlth Rep., Wash.*, 56, 1905.
Gwatkin, R. & Moore, T. (1940) *Canad. J. comp. Med.*, 4, 78.
Hammon, W. McD. *et al.* (1941) *Science*, 94, 328.
Hammon, W. McD., Reeves, W. C. & Sather, G. E. (1951) *J. Immunol.*, 67, 357.
Holden, P., (1955) *Proc. Soc. exp. Biol., N. Y.*, 88, 490.
Kissling, R. E. *et al.* (1951) *Proc. Soc. exp. Biol., N. Y.*, 77, 398.
Kissling, R. E. *et al.* (1957) *Amer. J. Hyg.*, 66, 48.
Marchetti, E. (1949) *Rev. Med. vet. B. Aires*, 31, 93.
Sooter, C. A. *et al.* (1951) *Proc. Soc. exp. Biol., N. Y.*, 77, 393.
Sooter, C. A., Howitt, B. F. & Gorrie, R. (1952) *Proc. Soc. exp. Biol., N. Y.*, 79, 507.
Ten Broeck, C., Hurst, E. W. & Traub, E. (1935) *J. exp. Med.*, 62, 677.
Tyzzer, E. E., Sellards, A. W. & Bennett, B. L. (1938) *Science*, 88, 505.

INFECTIOUS PORCINE ENCEPHALOMYELITIS

Babik, J. (1950) *Cas. Ceskoslovensk. Vet.*, 5, 326.

LOUPING ILL

Darling, F. F. (1947) *Natural history in the highlands and islands*. London, Collins, p. 111.
Dunn, A. M. (1960) *Brit. vet. J.*, 116, 284.

RUSSIAN SPRING-SUMMER ENCEPHALITIS

Chumakov, M. P., Vorobieva, N. N. & Sofronova, N. E. (1940) *Arch. Sci. biol., St. Pétersb.*, 59, 86.
Soloviev, V. D. (1941) *Trud. Venn., Med. Akad. Krasn. Arm.*, 25, 95.

COXSACKIE VIRUS INFECTION

Kraft, L. M. (1952) *Proc. Soc. exp. Biol., N. Y.*, 80, 498.
O'Connor, J. R. & Morris, J. A. (1954) *Fed. Proc.*, 13, 507.
Tobin, J. O'H. (1953) *Brit. med. Bull.*, 9, 201.

Avian encephalomyelitis

Jennings, A. R. (1954) *J. comp. Path.*, 64, 356.

Epizootic encephalitis in wild ducks in the United States

Rosenow, E. C. (1943) *Cornell Vet.*, 33, 277.

Murray valley encephalitis (Australian X disease)

Anderson, S. G. (1953) *Med. J. Aust.*, 25 April, 573.
Anderson, S. G. (1954) *J. Hyg.*, 52, 447.
Miles, J. A. R. & Howes, D. W. (1953) *Med. J. Aust.*, 1, 7.

Infectious encephalitis in foxes

Bezdek, H. (1942) *J. Mammal.*, 23, 98.
Green, R. G. *et al.* (1930) *Amer. J. Hyg.*, 12, 109.
Green, R. G., Evans, C. A. & Yanamura, H. Y. (1943) *Amer. J. Hyg.*, 53, 186.

Bilirubinemia and jaundice in raccoons

Kilham, L. & Herman, C. M. (1954) *Proc. Soc. exp. Biol., N. Y.*, 85, 272.

Dengue

Rowan, L. C. & O'Connor, J. L. (1957) *Nature*, 179, 786.

Japanese encephalitis

Anderson, S. G. (1953) *Med. J. Aust.*, 25 April, 573.
Anderson, S. G. (1954) *J. Hyg.*, 52, 447.
Tabuchi, E., Hosoda, T. & Narita, R. (1951) *2nd Rep. Jap. eq. Enceph.*, 93.

Diseases due to protozoa and closely allied organisms

Leishmaniasis

Ansari, N. & Faghih, M. (1953) *Ann. Parasit. hum. comp.*, 28, 241.
Del Ponte, E. (1952) *Bol. Ofic. sanit. Pan-amer.*, 32, 223.
Heisch, R. B. (1954) *Trans. R. Soc. trop. Med. Hyg.*, 48, 449.
Hoare, C. A. (1955) *Vet. Rev.*, 1, part 2, 62.
Kojevnikov, P. V. (1941) *Symp. 1st Int.-Repub. Conf. on Cutaneous Leishmaniasis and Sandflies, Ashkhabad*, 127.
Kojevnikov, P. V. (1942) *Cutaneous leishmaniasis.* Ashkhabad.
Latyshev, N. I. & Kriukova, A. P. (1942) *Med. Parasit., Moscow*, 11, Nos. 1 and 2, p. 74.
Latyshev, N. I., Kriukova, A. P. & Pvalishina, T. P. (1951) *Problems of regional, general and experimental parasitology, Moscow*, 7, 35.

Trypanosomiasis (sleeping sickness and Chagas' disease)

Corson, J. F. (1936) *Trans. Roy. Soc. trop. Med. Hyg.*, 29, 690.
Duke, H. L. (1936) *Trans. Roy. Soc. trop. med. Hyg.*, 30, 129.
Fairbairn, H. (1948) *Trop. Dis. Bull.*, 45, 1.
Floch, H. & Fauran, P. (1954) *Ann. Parasit. hum. comp.*, 29, 499.
Hoare, C. A. (1948) *Proc. R. Soc. Med.*, 41, 553.
Hoare, C. A. (1953) *Trans. R. Soc. trop. Med. Hyg.*, 47, 271.
Hoare, C. A. (1955) *Vet. Rev.*, 1, part 2, 62.
Pifano, C. F. (1954) *Arch. Venezol. Patol. trop. Parasit. med.*, 2, 89.
Woody, N. C. & Woody, H. B. (1955) *J. Amer. med. Ass.*, 159, 676.
Zeledan, R. (1954) *Rev. Biol. trop. Costa Rica.*, 2, 231.

Trypanosomiasis in game animals

Buxton, P. A. (1955) *The natural history of tsetse flies.* London, Lewis.
Bruce, D. *et al.* (1914) *Rep. Sleep. Sickn. Comm. roy. Soc.*, 15, 16.
Buchanan, J. R. C. (1929) *Kenya E. Afr. med. J.*, 6, 111.
Potts, W. H. & Jackson, C. H. N. (1952) *Bull. ent. Res.*, 43, 365.
Rangel (1905) *Bol. Lab. Hosp. Vargas.* (*Caracas*), 2, 11.
Vanderplank, F. L. (1942) *Trans. R. Soc. trop. Med. Hyg.*, 35, 319.
Vanderplank, F. L. (1947) *Ann. trop. Med. Parasit.*, 41, 365.
Weitz, B. & Jackson, C. H. N. (1955) *Bull. ent. Res.*, 46, 531.
Wenyon, C. M. (1926) *Protozoology*, London, Baillière, Tindall and Cox.

Other evidence of trypanosomiasis in wildlife

Heisch, R. B. (1952) *Nature, Lond.*, 169, 118.
Werner, H. (1949) *Zbl. Bakt.*, 154, 68.

Trichomoniasis

Callender, G. R. & Simmons, J. S. (1937) *Amer. J. trop. Med.*, 17, 124.
Cauthen, G. E. (1936) *Amer. J. Hyg.*, 23, 132.
Haugen, A. O. (1952) *J. Wildlife Mgmt.*, 16, 164.
McNeil, E., Platt, A. E. & Hinshaw, W. R. (1939) *Cornell Vet.*, 29, 330.
Schoop, G. & Stolz, A. (1939) *Dtsch. tierärztl. Wschr.*, 47, 113.
Stabler, R. M. (1937) *J. Parasit.*, 23, 554.

Histomoniasis (blackhead)

Hagan, W. A. (1943) *The infectious diseases of domestic animals.* New York, Comstock.
Salhoff, S. (1938) *Berl. tierärztl. Wschr.*, 28 Jan., 49.
Wenrich, D. H. (1941) *J. Parasit.*, 27, Suppl., 27.
Wilson, J. E. (1952) Personal communication.

Coccidiosis

Boughton, D. C. (1937) *Amer. J. Hyg.*, 25, 203.
Brinkmann, A. (1926) *Bergens Museums Aarbok*, p. 3.
Carvalho, J. C. M. (1943) *Iowa St. Coll. J. Sci.*, 18, 103.
Clapham, P. (1954) *Vet. Rec.*, 66, 100.
Herman, C. M., Jankiewicz, H. A. & Saarni, R. W. (1942) *Condor*, 44, 168.
Honess, R. F. (1939) *J. Parasit.*, 31, 281.
Honess, R. F. (1942) *Bull. Wyo. agric. Exp. Sta.* No. 249, p. 28.
Levine, N. D. (1953) *Amer. Midl. Nat.*, 49, 696.
Richardson, U. F. (1948) *Veterinary protozoology.* Edinburgh, Oliver and Boyd.
Ritchie, J. (1926) *Trans. Perthsh. Soc. nat. Sci.*, 8, 156.
Salhoff, S. (1939) *Berl. Münch. tierärztl. Wschr.*, 25 August, 537.
Smith & Smillie (1917) *J. exp. Med.*, 25, 415.
Yakimoff, W. L. & Matschoulsky, S. N. (1935) *Arch. Inst. biol.* (Def. agric. anim.), *S. Paulo*, 6, 171.
Yakimoff, W. L., Matschoulsky, S. N. & Spartansky, O. A. (1939) *J. R. micr. Soc.*, 59, 30.

Hemoproteus, Leucocytozoon, Plasmodium

Beltran, E. (1940) *Ciencia Mex.*, 1, 20.
Borg, K. (1953) *Monograph on Leucocytozoan*; *Stu. vet. med. Inst. Stockholm.*
Clark, C. H. D. (1934) *Science*, 80, 228.
Clark, C. H. D. (1935a) *Canad. J. Res.*, 12, 646.
Clark, C. H. D. (1935b) *Trans. 21st Amer. Game Conf.*, 402.
Coatney, G. R. (1937) *J. Parasit.*, 23, 556.
Coles, A. C. (1914) *Parasitology*, 7, 17.
Enigk, K. (1941) *Dtsch. tierärtzl. Wschr.*, 50, 177.
Gaud, J. & Petitot, M. L. (1945) *Arch. Inst. Pasteur, Maroc*, 3, 149.
Herman, C. M. (1935) *Incidence of blood parasites in birds.* New York, Syracuse University. (Thesis)
Herman, C. M. (1937) *J. Parasit.*, 23, 553.
Herman, C. M. (1944) *Bird Banding*, 15, 3: 89.
Herman, C. M. *et al.* (1954) *Amer. J. trop. Med.*, 3, 676.
Hewitt, R. (1940) *J. Parasit.*, 26, 287.
Huff, C. G. (1939) *J. Amer. vet. med. Ass.*, 94, 615.
Jordan, H. B. (1943) *J. Parasit.*, 29, 260.
Manwell, R. D. (1938) *Amer. J. trop. Med.*, 18, 565.
Morello, E. (1938) *Profilassi*, 11, 175.
Nelson, E. C. & Gashwiler (1941) *J. Wildlife Mgmt*, 5, 199.
O'Roke, E. C. (1930) *J. Parasit.*, 17, 2: 112.
O'Roke, E. C. (1931) *J. Parasit.*, 18, 127.
O'Roke, E. C. (1934) *Univ. Mich. School of Forestry and Cons. Bull.* No. 4.
Papadakis, A. (1935) *Prakt. Akad. Athen.*, 10, 432.
Rodhain, J. & Andrianne, V. (1953) *Ann. Parasit. hum. comp.*, 27, 573.
Thompson, P. E. (1943) *J. Parasit.*, 29, 153.

Wetmore, P. W. (1939) *J. Wildlife Mgmt*, 3, 361.
Wetmore, P. W. (1941) *J. Parasit.*, 27, 379.

THEILERIOSIS

Neitz, W. O., Canham, A. S. & Kluge, E. B. (1955) *J. S. Afr. vet. med. Ass.*, 26, 79-87.
Richardson, U. F. (1948) *Veterinary protozoology*. Edinburgh, Oliver and Boyd, p. 60.
Seddon, H. R. (1952) *Diseases of domestic animals in Australia*. Part 4. Australia, Department of Health (Division of Veterinary Hygiene). Service Publication No. 8.

PIROPLASMOSIS AND ANAPLASMOSIS

Boynton, W. H. & Woods, G. M. (1940) *Science*, 91, 168.
Heisch, R. B. (1952) *Ann. trop. Med. Parasit.*, 46, 150.
Jansen, B. C. (1952) *Onderstepoort J. vet. Sci.*, 25 (3), 3.
Neitz, W. O. (1938) *Onderstepoort J. vet. Sci.*, 10, 37.
Osebold, J. W., *et al.* (1959) *Cornell Vet.*, 49, 97.

TOXOPLASMOSIS

Beverley, J. K. A., Beattie, C. P. & Roseman, C. (1954) *J. Hyg.*, 52, 37.
Borg, K. (1953a) *Monograph on Leucocytozoan: Stat. Vet. Med. Inst. Stockholm.*
Borg, K. (1953b) *Proc. 15th Int. vet. Congress, Stockholm*, 2, 90.
Christiansen, M. (1948) *Medlemsbl. Danske Dyrlaegeforen*, 31, 1.
Findlay, G. M. & Middleton, A. D. (1934) *J. Anim. Ecol.*, 3, 150.
Herman, C. M. (1937) *Amer. J. Hyg.*, 25, 303.
Hülphers, G., Lillengen, K. & Rubarth, S. (1947) *Svensk VetTidskr.*, 52, 295.
Jacobs, L., Melton, M. L. & Jones. F. E. (1952) *J. Parasit.*, 38, 457.
Manwell, R. D. & Drobeck, H. P. (1951) *Exp. Parasit.*, 1, 83.
Nicolle & Manceaux (1909) *C. R. Acad. Sci., Paris*, 148, 569.
Studić, D. (1953) *Veterinaria, Sarajevo*, 2, 352.
Weinman, D. & Chandler, A. H. (1954) *Proc. Soc. exp. Biol., N. Y.*, 87, 211.

SARCOSPORIDIOSIS

Darling (1910) *J. exp. Med.*, 12, 19.
Farris, E. J. & Griffith, J. Q. (1949) In, *The rat in laboratory investigation*. 2nd rev. ed. Philadelphia, Lippincott.
Hagan, W. A. (1943) *Infectious diseases of domestic animals*. New York, Comstock.
Quortrup, E. R. & Shillinger, J. E. (1941) *J. Amer. vet. med. Ass.*, 99, 382.
Quortrup, E. R. & Sudheimer, R. L. (1944) *J. Amer. vet. med. Ass.*, 104, 29.
Scott, J. W. (1943a) *Bull. Wyo. Agric. Exp. Sta.* No. 259.
Scott, J. W. (1943b) *Bull. Wyo. Agric. Exp. Sta.* No. 262.
Shillinger, J. E. & Wetmore, P. W. (1938) *Trans. 3rd Amer. Wildlife Conf. Wash.*, p. 898. American Wildlife Institute.
Wickware, A. B. (1944) *Canad. J. comp. Med.*, 8, 15.

Diseases due to rickettsiae

Mexican typhus

Brumpt, E. (1932) *Bull. Acad. Méd., Paris*, 107, 356.
Ceder, E. T. *et al.* (1931) *Publ. Hlth Rep., Wash.*, 46, 3103.
Dyer, R. E., Rumreich, A. & Badger, L. F. (1931a) *Publ. Hlth Rep., Wash.*, 46, 334.
Dyer, R. E., Rumreich, A. & Badger, L. F. (1931b) *J. Amer. med. Ass.*, 97, 589.
Dyer, R. E. *et al.* (1931c) *Publ. Hlth Rep., Wash.*, 46, 1869, 2415.
Dyer, R. E. *et al.* (1932a) *J. infect. Dis.*, 51, 137.
Dyer, R. E. *et al.* (1932b) *Publ. Hlth Rep., Wash.*, 47, 931.
Dyer, R. E. *et al.* (1932c) *Publ. Hlth Rep., Wash.*, 47, 987.
Kodama, M. & Takashashi, K. (1931) *Zbl. Bakt.*, 119, 311.
Kodama, M., Kono, M. & Takashashi, K. (1932) *Kitasato Arch. exp. Med.*, 9, 91.
Kritschewski, I. L. & Solowiow, N. N. (1934) *Zbl. Bakt.*, 131, 232.
Lawton, F. B. & Murray, A. (1933) *Med. J. Aust.*, 20, part 1, 773.
Lépine, P. (1932) *C. R. Acad. Sci., Paris*, 194, 401.
Marchandier *et al.* (1931) *Bull. Acad. Méd., Paris*, 105, 1012.
Maxcy, K. F. (1926) *Publ. Hlth Rep., Wash.*, 41, 1213, 2967.
Maxcy, K. F. (1928) *Publ. Hlth Rep., Wash.*, 43, 3084.
Maxcy, K. F. (1929) *Publ. Hlth Rep., Wash.*, 44, 589, 1735, 1935.
Mooser, H. (1928) *J. infect. Dis.*, 43, 261.
Mooser, H., Castaneda, M. R. & Zinsser, H. (1931) *J. Amer. med. Ass.*, 97, 231.
Nicolle, C. & Sparrow, H. (1934) *Arch. Inst. Pasteur, Tunis*, 23, 247.
Penfold, W. J. & Corkill, A. B. (1928) *Med. J. Aust.*, 15, part II, 304.
Rumreich, A. S. (1933) *J. Amer. med. Ass.*, 100, 331.
Suzuki, K. (1934) *Zbl. Bakt.*, 131, 236.

Japanese flood fever (tsutsugamushi)

Lewthwaite, R. & Savoor, S. R. (1936) *Trans. R. Soc. trop. Med. Hyg.*, 29, 56.
Sellards, A. W. (1923) *Amer. J. trop. Med.*, 3, 529.

Rocky Mountain spotted fever

Fukuda, Y. (1929) *Zbl. Bakt.*, 111, 408.

Trench fever

Braslawsky, P. I. (1934) *Münch. med. Wschr.*, 81, 172.
Laurell, A. (1933) *Klin. Wschr.*, 12, 713.

Q fever

Babudieri, B. & Moscovici, C. (1952) *Nature, Lond.*, 169, 195.
Derrick, E. H. *et al.* (1939) *Med. J. Aust.*, Jan. 28, 150.
Derrick, E. H. & Smith, D. J. W. (1940) *Aust. J. exp. Biol. med. Sci.*, 18, 99.
Pope, J. H., Scott, W. & Dwyer, R. (1960) *Aust. J. exp. Biol. med. Sci.*, 38, 17.

Rickettsial pox

Huebner, R. J. (1950) *Bact. Rev.*, 14, 245.

Heartwater

Neitz, W. O. (1935) *Onderstepoort J. vet. Sci.*, 5, 35.
Neitz, W. O. (1944) *Onderstepoort J. vet. Sci.*, 20, 25.

Diseases due to true neoplasms and viruses associated with tumor formation

Tumors of deer

Bouvier, G., Burgisser, H. & Schneider, P. A. (1955) *Schweiz. Arch. Tierheilk.*, 97, 323.
Chaddock, T. T. (1939) *Wis. Conserv. Bull.*, 4, 31.
Cowan, I. McT. (1951) *Proc. 5th Annual Game Convent., Victoria, B. C.*, p. 61.
Herman, C. M. & Bischoff, A. I. (1950) *Calif. Fish Game*, 36, 19.
Krause, C. (1938) *Arch. wiss. prakt. Tierheilk.*, 73, 1.
Krembs, J. (1939) *Tierärztl. Rdsch.*, 45, 763, 773.
Kumer, L. (1935) *Wien. klin. Wschr.*, 48, 890.
Muroma, E. (1954) *Suomen Riista*, 9, 186.
Quortrup, E. R. (1946) *Virginia Wild Life*, 7, 15.
Runge, S. & Witkowski, B. (1936) *Wiad. weteryn.*, 15, 232.
Shope, R. E. (1955) *Proc. Soc. exp. Biol., N. Y.*, 88, 533.
Stroh, G. (1937) *Berl. Tierärztl. Wschr.*, 14 May, 305.
Wadsworth, J. R. (1954) *J. Amer. vet. med. Ass.*, 124, 194.

Transmissible tumors of wild rabbits

Papillomatosis

Kidd, J. G. & Rous, P. (1940) *J. exp. Med.*, 71, 469.
Shope, R. E. & Hurst, E. W. (1933) *J. exp. Med.*, 58, 607.

Shope fibroma

Shope, R. E. (1932) *J. exp. Med.*, 56, 793, 803.

Myxomatosis

Aragao, H. de B. (1943) *Mem. Institut. Osw. Cruz.*, 38, 93.
Bull, L. B. & Mules, M. W. (1944) *J. Coun. sci. industr. Res. Aust.*, 17, 79.
Bull, L. B., Ratcliffe, F. N. & Edgar, G. (1953) *Proc. 15th Int. vet. Congress*, 1.
Fenner, F. & Marshall, I. D. (1954) *J. Hyg.*, 52, 321.
Findlay, G. M. (1929) *Brit. J. Exp. Path.*, 10, 214.
Hudson, J. R. & Mansi, W. (1955) *Vet. Rec.*, 67, 747.

Hurst, E. W. (1937) *Brit. J. exp. Path.*, 18, 1.
Jacotot, H. (1953) *Bull. Acad. Agric. de France*. 15 April. Monograph. Myxomatosis.
Jacotot, H. & Vallée, A. (1953) *Ann. Inst. Pasteur*, 85, 133.
Jacotot, H., Vallée, A. & Virat, B. (1953a) *Bull. Acad. vét. Fr.*, 26, 431.
Jacotot, H., Vallée, A. & Virat, B. (1953b) *Ann. Inst. Pasteur*, 85, 801.
Jacotot, H., Vallée, A. & Virat, B. (1954a) *Acad. d'Agric. France*, 28 Oct.
Jacotot, H., Vallée, A. & Virat, B. (1954b) *Ann. Inst. Pasteur*, 86, 374.
Jacotot, H., Vallée, A. & Virat, B. (1954c) *Bull. Acad. vét. Fr.*, 27, 465.
Jacotot, H., Vallée, A. & Virat, B. (1955a) *Ann. Inst. Pasteur*, 88, 234.
Jacotot, H., Vallée, A. & Virat, B. (1955b) *Ann. Inst. Pasteur*, 88, 1.
Jacotot, H., Vallée, A. & Virat, B. (1955c) *Ann. Inst. Pasteur*, 88, 381.
Jacotot, H., Vallée, A. & Virat, B. (1955d) *Ann. Inst. Pasteur*, 89, 8.
Jacotot, H. *et al.* (1954) *Ann. Inst. Pasteur*, 87, 477.
Lapage, G. (1954) *Nature*, 173, 856.
Lockley, R. M. (1955) *Nature*, 175, 906.
Myers, K., Marshall, I. D. & Fenner, F. (1954) *J. Hyg.*, 52, 337.
Rivers, T. M. (1927) *Proc. Soc. exp. Biol., N. Y.*, 24, 435.
Rivers, T. M. (1930) *J. exp. Med.*, 51, 965.
Sanarelli, G. (1898) *Zbl. Bakt.*, 23, 865.
Shanks, P. L. *et al.* (1955) *Brit. vet. J.*, 111, 25.
Thomson, H. V. (1954) *Agriculture, Lond.*, 61, 317.

Tumors in whales

Cockrill, W. Ross. (1960) *Brit. vet. J.*, 116.
Rewell, R. E. & Willis, R. A. (1949) *J. Path. Bact.*, 61, 454.
Rewell, R. E. & Willis, R. A. (1950) *J. Path. Bact.*, 62, 450.
Stolk, A. (1952) *Kon. Ned. Akad. Weten. Amsterdam. Proc. Series* C. 55, No. 3, 275.

Tumors in other wild mammals

Bouvier, G., Burgisser, H. & Schneider, P. A. (1954) *Monographie des maladies du lièvre en Suisse*. Lausanne, Service vétérinaire cantonal et Institut Galli-Valerio, p. 19.
Cheatum, E. L. (1951) *Cornell Vet.*, 41, 136.
Figge, F. H. J. (1952) *Cancer Res.*, 12, 261.
Habermann, R. T., Williams, F. P. & Eyestone, W. H. (1954) *J. Amer. vet. med. Ass.*, 125, 295.
Kilham, L., Herman, C. M. & Fisher, E. R. (1953) *Proc. Soc. exp. Biol., N. Y.*, 82, 298.
Zoller, H. (1940) *Dtsch. tierärztl. Wschr.*, 48, 142.

Tumors in birds

Clapham, P. Personal communication.
Cowan, I. McT. (1940) *J. Wildlife Mgmt*, 4, 311.
Jennings, A. R. (1954) *J. Comp. Path.*, 64, 356.
Krahnert, R. (1952) *Mh. VetMed.*, 7, 71.

ADDITIONAL REFERENCES

Cockrill, W. Ross. (1960) *Brit. Vet. J.*, 116, Nos. 4-5. (Pathology of the Cetacea, a veterinary study on whales)

Halloran, Patricia O'C. (1955) *Amer. J. vet. Res.*, 16. (A bibliography on diseases, anatomy, management, nutrition and other matters concerning wild animals in captivity and also in the free-living state)

Herman, C. M. (1955) *Recent studies in avian biology*. Urbana, University of Illinois Press. (A general treatise on diseases of birds)

Keymer, I. F. (1958) *Vet. Rec.*, 70, 713, 736. (Two general articles on causes of morbidity and mortality in wild birds)

Williams, M. C. (1957) *Vet. Rec.*, 99, 303. (Birds in relation to the arthropod-borne virus zoonoses)

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